peds
cases

ENURESIS: COUNSELLING & MANAGEMENT

INITIAL MANAGEMENT

1. Determine if…
- ☐ **Both** child and parent see enuresis as problematic, and are **motivated** to participate in treatment
- ☐ The child is **mature** enough to engage in and assume responsibility for treatment

2. Treat co-existing conditions:
- ☐ **Constipation**, sleep disordered breathing, ADHD, underlying stressors, poor self-concept, psychologic

3. Educate, emphasizing…
- ☐ **High prevalence** and generally **self-resolving** natural history
- ☐ Child should **NOT** be punished for bedwetting
- ☐ Usefulness of bed protection, absorbent undergarments, room deodorizers
- ☐ Avoiding sugary and caffeinated beverages

4. Establish goals and expectations:
- ☐ Determine family **priorities** (Reassurance? Staying dry for sleepovers? Decreasing # wet nights?)
- ☐ May involve several methods, be prolonged, fail in short term, often relapses
- ☐ **Slow, steady improvement** is more realistic

Voiding Diary

Should record:
- **Time** of void
- **Volume** voided
- **Relationship** to events (meals, school recess, play activities, stress)
- Episodes of **urgency** or **incontinence**

Personalized Calendar

Record:
- Daytime incontinence
- Enuresis events
- Encopresis
- Frequency & timing of bowel movements

- Helps to follow progress
- Parents should be cautious of implementing a reward system
- **AVOID** punishment and humiliation

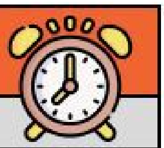
BEHAVIOURAL THERAPY

Goal: **achieve good bladder and bowel habits**

- ✓ Encourage **frequent voids**
  - Introduce timed voiding **every 2 hours**, regardless of if child feels the need to void
  - Avoid holding urine, urgency, and incontinence
  - Ensure easy access to toilets at school & home
  - Always have child void immediately before sleep
- ✓ Encourage **daily bowel movements**
  - Establish a schedule at specific time of day such as after breakfast before leaving for school
  - PEG 3350 for constipation
- ✓ Consume **majority of fluids in morning** and afternoon, minimize after dinner
- ✓ Encourage **physical activity** and discourage prolonged sitting
- ✓ Requires supportive environment, child motivation, patience, and time (average 6 months)

ACTIVE THERAPY

Similar outcomes, choose based on patient preference and fit with family



Pharmacologic Therapy

- **Desmopressin:**

Goal: optimize oral medication to **reduce production of urine overnight** (ADH analogue).
- ➢ Take medication **60 minutes** before bedtime
- ➢ **No fluid intake** 1 hour prior to and 8 hours after taking medication
- ➢ WATCH FOR: signs of symptomatic hyponatremia with water intoxication: discontinue if developing headache, nausea, vomiting.

Can be used **intermittently**, thus a good option for **special occasions** such as sleepovers and camps.

- **Anticholinergics and tricyclic agents** (second and third line): may be considered if other therapeutic options have failed.

Bed Alarms

Goal: teach child to awaken from sensation of a full bladder. Sensors attached to child's undergarments are connected to an **alarm** that **awakens the child at the moment of bed wetting**.
- ➢ Should be using **every night**
- ➢ Initially, child may not awaken from alarm, requiring parent to awaken child instead
- ➢ Child should then void in the **washroom**
- ➢ Return to sleep

- Most effective in children **>7 years old**
- Generally see **initial response in 1 – 2 months**
- **3 – 4 month** trial of continuous therapy is recommended
- Discontinue when dry for 14 consecutive nights, or if no improvement at one month
- Effective long term in **< 50%** of children
- **Recommend for older, motivated children from cooperative families**

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Sarah Park (Medical Student C2022, University of Alberta), Dr. Peter Metcalfe (Pediatric Urologist, University of Alberta) for www.pedscases.com

ACUTE ABDOMINAL PAIN

BACKGROUND

- Acute abdominal pain is a **common** pediatric presentation.
- It accounts for **~10% of primary care visits** and **~10% of emergency department (ED) visits**.
- Abdominal pain can be **visceral**, **somatic**, or **referred**.
- Much of the time, abdominal pain is **self-resolving** and **non-life-threatening**.
- In children, **constipation** is the most frequently identified cause of acute abdominal pain.
- Between **10-30%** of pediatric ED visits for acute abdominal pain require **surgical intervention**.
- **Appendicitis** is the most common surgical emergency.

HISTORY

- **HPI:** *OPQRST* [onset (gradual vs. sudden), palliating/provoking factors, quality, region & radiation, severity, timing (intermittent vs. constant)], previous episodes of similar pain, trauma history, pain interfering with activities or sleep
- **Associated symptoms**: fever, vomiting (bilious vs. non-bilious, bloody), hard stools, diarrhea, bloody stool, anorexia, cough, SOB, sore throat, urinary symptoms, vaginal bleeding/discharge, joint pain, rash, weight loss
- **Past medical & past surgical history**
- **Menstrual history**: age at menarche, duration, frequency, blood flow, dysmenorrhea, last menstrual period (LMP)
- **Sexual history**: partners, practices, past sexually transmitted infections (STIs), STI protection, contraception
- **Medication history**: some meds can cause nausea or abdominal pain
- **Family history**: sickle cell anemia, cystic fibrosis, etc.

Clinical pearl: if pain precedes vomiting, it is likely a surgical cause; however, if the *pain follows vomiting*, it is more likely to be a *medical cause*.

PHYSICAL EXAM

- **General appearance & vitals**: *sick vs. not sick*, activity level, food and fluid intake, fluid status, jaundice, etc.
- **ENT exam**: otitis media, pharyngeal exudate or erythema
- **Pulmonary exam**: focal consolidation
- **Abdominal exam**: bowel sounds, palpation for enlarged organs or masses, distention, guarding, rebound tenderness, Murphy's sign, Rovsing's sign, iliopsoas and obturator tests, McBurney's point tenderness
- **+/- DRE**: presence of constipation (hard stool in the rectal vault), melena
- **+/- Testicular exam**
- **+/- Pelvic exam**: pubertal girls, discharge, cervical motion tenderness, etc.

INVESTIGATIONS

Initial investigations:	Other investigations to consider depending on the history and presentation:
CBCdiff CRP Urinalysis β-hCG	Lipase, amylase LFTs Chlamydia & gonorrhea Stool studies Ultrasonography Abdominal x-ray Upper GI study CT or MRI

However, not all children presenting with acute abdominal pain require investigations. Investigations should be guided based on history and physical exam.

DIFFERENTIAL DIAGNOSIS FOR ACUTE ABDOMINAL PAIN IN PEDIATRICS

➢ Considering a differential diagnosis by **age group** is a logical approach as the differential for acute abdominal pain is vast.
➢ Before you even step into the room to meet the patient, you can have an idea of potential diagnoses based on age.

INFANTS & TODDLERS (0-4 years)

- Colic
- Cow's milk protein allergy
- Hirschsprung disease
- Incarcerated hernia
- Intussusception
- Lead poisoning
- Malrotation of the midgut
- Meckel diverticulum
- Necrotizing enterocolitis

SCHOOL AGE (5-11 years)

- Abdominal migraine
- Diabetic ketoacidosis
- Functional pain
- Henoch-Schonlein purpura
- Lactose intolerance
- Lead poisoning
- Mononucleosis
- Perforated ulcer

ADOLESCENCE (12-18 years)

- Diabetic ketoacidosis
- Dysmenorrhea
- Early pregnancy loss
- Ectopic pregnancy
- Functional pain
- Henoch-Schonlein purpura
- Hepatitis
- Inflammatory bowel disease
- Irritable bowel syndrome
- Lactose intolerance
- Mononucleosis
- Pelvic inflammatory disease
- Perforated ulcer
- Ruptured ovarian cyst
- Sexually transmitted infections
- Urolithiasis

ALL AGES (0-18 years)

- Adhesions
- **Appendicitis**
- Bowel obstruction
- **Child abuse**
- **Constipation**
- Dietary allergies
- Gallbladder disease
- **Gastroenteritis**
- Hemolytic uremic syndrome
- Hepatitis
- Mesenteric adenitis
- Myocarditis/pericarditis
- Ovarian torsion
- Pancreatitis
- Sickle cell crisis
- Testicular torsion
- Trauma
- Tumour
- Urinary tract infection
- Viral infection
- Volvulus

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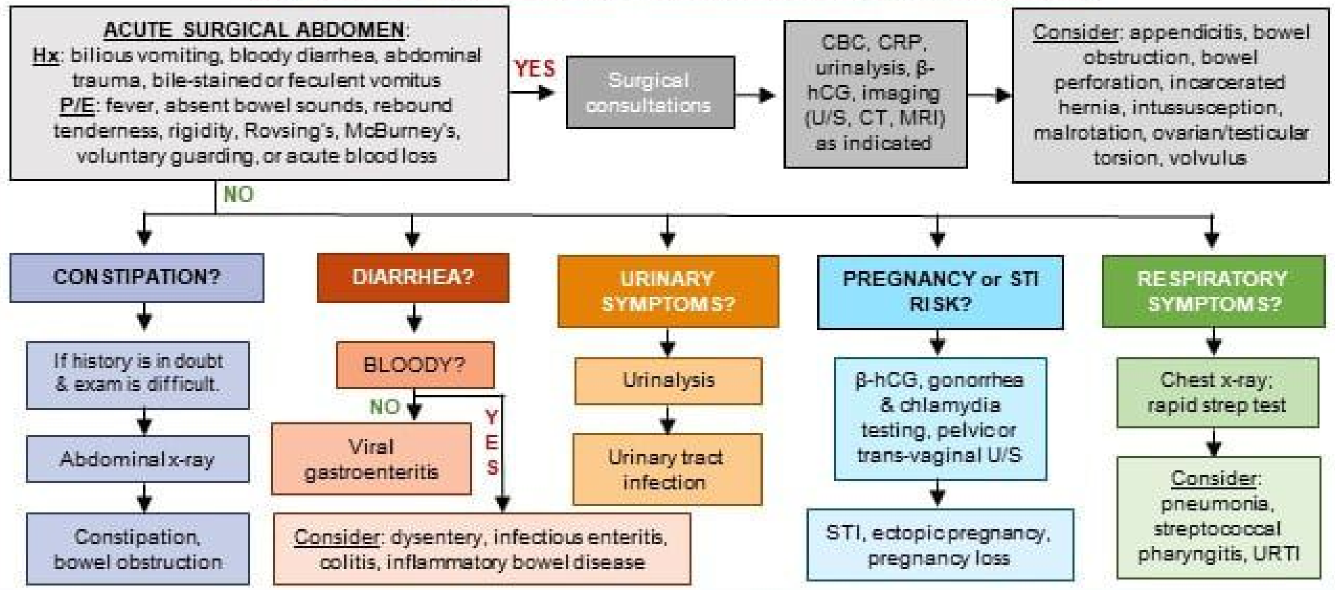
Katharine V. Jensen (Medical Student 2021, University of Alberta), Dr. Peter MacPherson (Pediatrician & Assistant Professor, Queen's University), & Dr. Melanie Lewis (Pediatrician & Professor, University of Alberta) for www.pedscases.com

ACUTE ABDOMINAL PAIN

EVALUATION & DIAGNOSTIC ALGORITHM FOR ACUTE ABDOMINAL PAIN



ACUTE SURGICAL ABDOMEN:
Hx: bilious vomiting, bloody diarrhea, abdominal trauma, bile-stained or feculent vomitus
P/E: fever, absent bowel sounds, rebound tenderness, rigidity, Rovsing's, McBurney's, voluntary guarding, or acute blood loss

YES → Surgical consultations → CBC, CRP, urinalysis, β-hCG, imaging (U/S, CT, MRI) as indicated → Consider: appendicitis, bowel obstruction, bowel perforation, incarcerated hernia, intussusception, malrotation, ovarian/testicular torsion, volvulus

NO →

CONSTIPATION? → If history is in doubt & exam is difficult. → Abdominal x-ray → Constipation, bowel obstruction

DIARRHEA? → BLOODY? → NO → Viral gastroenteritis; YES → Consider: dysentery, infectious enteritis, colitis, inflammatory bowel disease

URINARY SYMPTOMS? → Urinalysis → Urinary tract infection

PREGNANCY or STI RISK? → β-hCG, gonorrhea & chlamydia testing, pelvic or trans-vaginal U/S → STI, ectopic pregnancy, pregnancy loss

RESPIRATORY SYMPTOMS? → Chest x-ray; rapid strep test → Consider: pneumonia, streptococcal pharyngitis, URTI

APPENDICITIS

Acute appendicitis is the **most common** pediatric surgical emergency.

PEDIATRIC APPENDICITIS SCORE (PAS)

CRITERIA	DESCRIPTION	SCORE
RLQ tenderness to percussion, hopping, or cough	No Yes	0 2
Migration of pain to RLQ	No Yes	0 1
Fever ≥ 38°C (100.4°F)	No Yes	0 1
Anorexia	No Yes	0 1
Nausea or vomiting	No Yes	0 1
Tenderness over right iliac fossa	No Yes	0 2
Leukocytosis WBC >10,000	No Yes	0 1
Neutrophilia ANC > 7,500	No Yes	0 1

MANAGEMENT BASED ON PAS SCORE

LOW RISK (< 4)	EQUIVOCAL (4-6)	HIGH RISK (> 6)
▪ Low likelihood of acute appendicitis ▪ Likely do not warrant imaging	▪ Imaging (U/S or MRI) ▪ Surgical consult *may* be warranted	▪ Surgical consult is warranted

Equivocal & high risk patients: maintain NPO, IV fluids, analgesia, and imaging or surgical consultation.

CONSTIPATION

The **most frequently identified** cause of acute abdominal pain is constipation.

CONSTIPATION

HISTORY	PHYSICAL EXAM
▪ Long history of **difficult or painful defecation** ▪ **Infrequent** bowel movements ▪ **Withholding behavior** ▪ **Poorly localized** pain, usually non-radiating	▪ Mild **tenderness** to palpation ▪ **Palpable fecal masses** ▪ DRE may reveal hard **stool in the rectal vault**

INVESTIGATIONS	MANAGEMENT
▪ Labs are usually unnecessary ▪ Consider **abdominal x-rays** if the history is in doubt and the physical exam is difficult.	▪ **Lactulose** or **polyethylene glycol 3350** PO or via NG tube ▪ Enemas are not recommended ▪ Maintenance regimen: **daily stool softeners**, daily timed **toilet sitting**, and **improved diet**

Functional constipation: (*most common*) does not have an anatomical or physiological cause, yet the child experiences distressing infrequent passage of uncomfortable and hard stools.

⚠ IMPORTANT POINTS ⚠

- ➢ Look for any **red flags** that would require further investigation and/or consultation.
- ➢ Remember that this may be an **acute presentation of a chronic problem** (e.g.: child presenting to the ED with abdominal pain from chronic constipation).
- ➢ If the child is being discharged with an uncertain diagnosis, it is crucial to inform families to **return to hospital** should **symptoms progress** or the child **becomes sicker**.

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ACUTE ASTHMA EXACERBATION

MANAGEMENT ALGORITHM

INITIAL ASSESSMENT

- **Vital signs, pulse oximetry**
- **Spirometry**: peak flows should be compared to normal values or the child's personal best, if known
- **Focused medical history**: previous meds, allergies, risk factors for ICU admission (previous ICU admissions, intubation, previous life-threatening asthma exacerbations, etc.)
- **Physical exam**: LOC, speech, activity, accessory muscle use, air entry, wheezing *(see previous page)*
- **Categorize disease severity**: Pediatric Respiratory Assessment Measure (PRAM) *(see previous page)*
- Ancillary tests, such as chest x-rays and blood gases, are not routinely recommended

THE SEVERITY OF THE ASTHMA EXACERBATION DETERMINES THE INITIAL TREATMENT (1 HOUR)

MILD

- Keep O_2 sats ≥ 94%
- Salbutamol q20 mins x 1-3 doses
- Consider inhaled corticosteroids (ICS)

MODERATE

- Keep O_2 sats ≥ 94%
- Salbutamol q20 mins x 3 doses
- Oral steroids (prednisone, prednisolone, dexamethasone)
- Consider ipratropium x 3 doses in 1 hour

SEVERE

- Keep O_2 sats ≥ 94%
- Consider 100% O_2
- Salbutamol and ipratropium x 3 doses
- Oral steroids
- Consider IV methylprednisolone, continuous aerosolized β2-agonist, IV $MgSO_4$
- Keep patient NPO

IMPENDING RESPIRATORY FAILURE

- Maintain O_2 sats ≥ 94%; non-rebreather mask with 100% O_2
- Continuous aerosolized salbutamol and ipratropium x 3 doses
- Keep patient NPO
- Establish IV assess
- Continuous cardiac and O_2 monitoring
- IV methylprednisolone
- Consider: IV $MgSO_4$, IV aminophylline, or IV salbutamol
- Blood gases, electrolytes
- Consider SC epinephrine
- If deteriorating, **call PICU** and consider rapid sequence intubation

Reassess the patient **1 hour after initial treatment & re-categorize severity**

Effective medical intervention decreases respiratory distress and improves oxygenation.

MILD

- Observe in the ED for 2 hours
- If no further treatment is required, continue ICS at home and follow-up with physician

MODERATE

- Keep O_2 sats ≥ 94%
- Salbutamol q1 hour
- Reassess in 2 hours

SEVERE

- Keep O_2 sats ≥ 94%
- Salbutamol q20 mins, until able to tolerate q1 hour
- Reassess frequently

IMPENDING RESP. FAILURE

- Same as above
- If deteriorating, **call PICU** and consider rapid sequence intubation

Reassess the patient & **re-categorize severity**. What is the severity after **2 hours** of treatment?

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- Reassess: if salbutamol is needed more than every 4 hours, **ADMIT TO HOSPITAL**

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EMERGENCY DEPARTMENT DISCHARGE CRITERIA

- Use of β2-agonist less often than q4 hours
- SpO_2 ≥ 94% on room air
- Improved air entry
- Minimal or no signs of respiratory distress

FOLLOW-UP PLAN

- Complete course of oral steroids
- Use of β2-agonist q4 hours, then prn
- Review inhaler techniques
- F/U with GP
- Referral to specialist if needed

May 2020
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A common, **chronic inflammatory** disorder of the airways characterized by **reversible airflow obstruction**, airway **hyper-responsiveness**, and **recurring symptoms** of wheezing, coughing, chest tightness, and shortness of breath.	A potentially life-threatening acute **worsening of symptoms** causing **significant distress** necessitating attention by healthcare professionals or administration of systemic corticosteroids.

- **Common triggers**: viral respiratory tract infections, suboptimal baseline control of asthma, and allergen exposures.
- Asthma exacerbations are the **leading cause of pediatric hospitalizations** and account for **3-7% of pediatric ED visits.**
- **50%** of children presenting to the ED with an asthma exacerbation are **< 5 years old.**

IMMEDIATE & OBJECTIVE ASSESSMENT OF THE ASTHMA EXACERBATION SEVERITY

CLINICAL FEATURES FOR THE DIFFERENT CLASSIFICATIONS OF ASTHMA SEVERITY

	MILD	MODERATE	SEVERE	IMPENDING RESP. FAILURE
MENTAL STATUS	Normal	May be agitated	Agitated	Drowsy, confused (signs of cerebral hypoxemia)
ACTIVITY & FEEDING	Normal activity, exertional dyspnea	↓ activity, ↓ feeding (infants)	↓ activity, stops feeding	Unable to feed
SPEECH	Normal speech	Speaks in **phrases**	Speaks in **words**	Unable to speak
WORK OF BREATHING	Minimal intercostal retractions	Intercostal and substernal retractions	Significant **respiratory distress**. Involves all accessory muscles, nasal flaring, paradoxical thoraco-abdominal movement.	Marked **respiratory distress** at rest. Involves all accessory muscles, nasal flaring, paradoxical thoraco-abdominal movement.
CHEST AUSCULTATION	Moderate wheeze	Pan-expiratory and inspiratory wheeze	Audible wheeze without stethoscope	Silent chest (no air entry), absence of wheeze
SpO_2 ON R/A	> 94%	91-94%	< 90%	< 90%
PEAK FLOW VS. PERSONAL BEST	> 80%	60-80%	< 60%	Unable to perform

PEDIATRIC RESPIRATORY ASSESSMENT MEASURE (PRAM) SCORING

Criteria	Description	Score	Important tips
O_2 saturation	≥ 95% 92-94% < 92%	0 1 2	The patient must be breathing **room air** for this measurement.
Suprasternal retractions	Absent Present	0 2	**Visual** assessment.
Scalene retractions	Absent Present	0 2	**Palpable** assessment, as scalenes are deep muscles that cannot be visualized.
Air entry	Normal ↓ at bases Widespread ↓ Minimal or absent	0 1 2 3	Use **lung fields** to grade air entry. If asymmetric, the rating is determined by the most severely affected field.
Wheezing	Absent Expiratory only Inspiratory ± expiratory Audible without stethoscope or silent chest (no air entry)	0 1 2 3	**≥ 2 auscultation zones** must be affected. If there is asymmetry, the rating is determined by the most severely affected zones.

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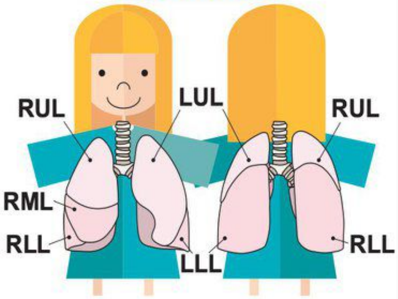

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Left posterior: LUL & LLL
Right anterior: RUL & RML
Right posterior: RUL & RLL

PRAM SCORE	SEVERITY
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4-7	MODERATE
8-12	SEVERE

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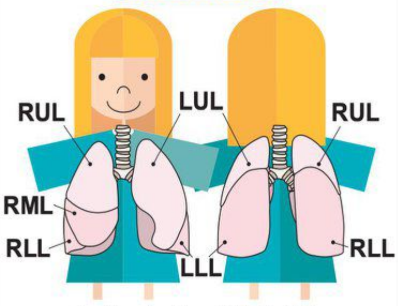

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ACUTE POST-INFECTIOUS CEREBELLAR ATAXIA

Acute Post-Infectious Cerebellar Ataxia *(aka. Post-Infectious Cerebellitis)* is the most common cause of acute cerebellar ataxia in children. It is a ***diagnosis of exclusion*** that requires a thorough H&P with appropriate choice of investigations.

PRESENTATION

HISTORY

- Typically, age < 6 years old
- Recent illness (1-3 weeks prior)
- Acute to subacute onset (hours – days)
- **Gait disturbance is often the primary concern**
- Otherwise well
- Developmentally & neurologically typical

PHYSICAL EXAM

General

- Alert & interactive
- Vitally stable

Neurologic *most common

- Wide based, staggering gait*
- Dysmetria*
- Dysdiadochokinesia*
- Truncal instability*
- Dysarthria
- End-gaze nystagmus
- Intention tremor
- Absence of extracerebellar abnormalities

DIFFERENTIAL DIAGNOSIS

DDx

- **Toxic ingestion**
- Infectious
  - Meningitis
  - Encephalitis
  - Acute cerebellitis
- Acute demyelinating encephalomyelitis (ADEM)
- Structural
  - Posterior fossa tumor
  - Trauma
  - Stroke
- Vertigo
- Inborn errors of metabolism

INVESTIGATIONS

- Toxicology screen
- Glucose
- Evaluate for current CNS or systemic infection based on presenting infectious signs & symptoms
  - Consider LP if atypical features or concern for CNS infection (may see lymphocytic pleocytosis)
- MRI should be considered (normal in most but cerebellar hyperintensities may be seen)

ETIOLOGY & PATHOPHYSIOLOGY

- Felt to be due to post-infectious autoimmune mediated inflammation or direct infection of cerebellum
- Systemic **viral infections** most common: EBV, Varicella
  - Others: COVID-19, HSV, coxsackie, measles, ERV, etc.
- Occasionally associated with bacteria

FEATURES SUGGESTIVE OF ALTERNATIVE DIAGNOSES

- Atypical age
- Lack of clinical improvement
- Altered level of consciousness
- Extreme irritability
- Signs of ↑ intracranial pressure
- Seizures
- Fever
- Meningismus
- Head trauma
- Focal or asymmetric neuro findings
- Insidious onset or prior episodes
- Developmental delay or regression
- Lack of prodromal infection

MANAGEMENT & OUTCOME

Typically, **self-limited** and symptoms improve over 2-3 weeks (complete recovery in 3 months)

- Supportive care with close follow up
- Consider a trial of pulse steroids (3-5 days) +/- IVIG if severe symptoms or lack of improvement
- Recurrence is rare

January 2025
Courtney Hebert (Medical Student, University of Alberta), Dr. Colin Wilbur (Pediatric Neurologist, Stollery Children's Hospital, University of Alberta) for www.pedscases.com

ACUTE RHINOSINUSITIS (ARS)

Rhinosinusitis is symptomatic inflammation of the nasal cavity and sinus. This common condition can be:

- Acute (<4 weeks)
- Subacute (4-12 weeks)
- Chronic (>12 weeks)
- Recurrent (4+ events of ARS in one year)

ETIOLOGY

Most causes of ARS are viral. Bacterial causes can be:

- *Streptococcus pneumoniae*
- *Haemophilus influenzae*
- *Moraxella catarrhalis*

PRESENTATION

Main symptoms of ARS (PODS):

- Facial **P**ain or pressure
- Nasal blockage/**O**bstruction
- Nasal purulence or post nasal discolored **D**ischarge
- Loss of **S**mell (hyposmia)

Non specific symptoms:

- Fatigue
- Headache
- Cough
- Fever
- Bad breath
- Poor sense of smell

PHYSICAL EXAM

- Tenderness of ethmoid and/or maxillary sinus
- Periorbital edema

Oral cavity examination

- palate integrity
- tenderness of maxillary teeth
- postnasal drip

Anterior rhinoscopy

- Mucosal edema
- Purulent drainage

COMPLICATIONS

Infection can spread beyond paranasal sinuses. This can cause:

- Orbital/periorbital cellulitis
- Orbital, epidural, subdural, or brain abscesses
- Cavernous sinus thrombosis

Look out for:

- Orbital edema, pain, proptosis or abnormal eye movement
- Decreased visual acuity: poor red-green colour differentiation or diplopia
- Sudden onset or severe headache
- Neck stiffness
- Cranial nerve palsy

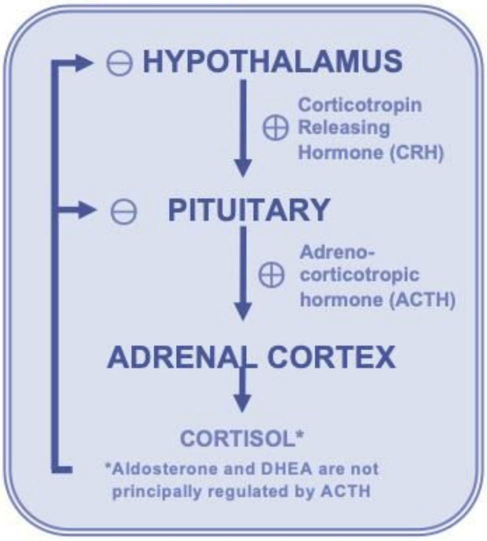
DIAGNOSIS

- ARS is a clinical diagnosis wherein the **PODS** symptoms may be present
- Investigations are usually unnecessary, but further investigation by imaging (CT/MRI) and microbial analysis can be indicated in the event of complications or lack of response to treatment
- ARS is suspected to be bacterial if symptoms remain for longer than 7 days and ≥ 2 of PODS are present

MANAGEMENT	**< 2 yrs old or complicated ARS**	**> 2 yrs old**
Viral	Symptomatic care Consider saline nasal irrigation or intranasal corticosteroids	
Bacterial	amoxicillin-clavulanate	amoxicillin
	Antibiotics are administered following 3 days observation Typical course is 10-14 days Obtain cultures to direct antibiotic therapy when possible	
Surgery can be an option should treatments fail or the patient has developed complications (see red box)		

April 2024
Divya Goel (Medical Student, University of Alberta), Dr. André Isaac (Pediatric Otolaryngologist, University of Alberta) for www.pedscases.com

ADDISON'S DISEASE



⊖ HYPOTHALAMUS

⊕ Corticotropin Releasing Hormone (CRH)

⊖ PITUITARY

⊕ Adreno-corticotropic hormone (ACTH)

ADRENAL CORTEX

CORTISOL*

*Aldosterone and DHEA are not principally regulated by ACTH

PATHOPHYSIOLOGY

- Acquired primary adrenal insufficiency
- Decreased production of adrenocortical hormones:
  - Glucocorticoid: Cortisol has a wide range of essential functions in the body including metabolism, immune, and blood pressure regulation
  - Mineralocorticoid: Aldosterone is primarily responsible for regulating fluid status via Na and K excretion
  - Androgen: DHEA

ETIOLOGY

- **Autoimmune destruction**
- Infection
- Adrenal hemorrhage/infarction
- Metastatic infiltration
- Drugs
- Infiltrative Disease

PRESENTATION

SYMPTOMS

- Fatigue
- Weakness
- Weight Loss
- Salt Craving
- Postural Dizziness
- Myalgia/Arthralgia
- Nausea & Vomiting
- Abdominal Pain

PHYSICAL EXAM

- Signs of dehydration
- Orthostatic hypotension
- Hyperpigmentation

INVESTIGATIONS & DIAGNOSIS

- Low AM Cortisol suggests adrenal (glucocorticoid) insufficiency
- Elevated ACTH indicates a primary cause
- **ACTH stimulation test with cosyntropin for definitive diagnosis**
  - An inadequate rise in cortisol indicates adrenal insufficiency – should typically rise to >500 nmol/L
- Elevated renin indicative of mineralocorticoid deficiency
- Electrolyte imbalance due to mineralocorticoid deficiency
  - K+: elevated
  - Na+: low to normal

MANAGEMENT

- Glucocorticoid replacement (e.g. hydrocortisone, prednisone)
  - Hydrocortisone (t.i.d. or q.i.d.) is the preferred choice as it more closely mimics natural production of cortisol and has less risk of growth suppression
  - Monitor for cortisol excess – poor growth, obesity, striae, hypertension, ↓ bone mineral density, and glucose intolerance
- Mineralocorticoid replacement (e.g. fludrocortisone) to maintain fluid and electrolyte balance
- Patient education with yearly review of sick days, emergency contact information, and written instructions

URGENT/IMPORTANT:

- Ensure patients are **stress dosing** their glucocorticoid appropriately:
  - **2-3x their normal dose when experiencing acute stress** – e.g. fever, vomiting, diarrhea
  - When under critical stress (e.g. sepsis, trauma, OR) stress dosing will be >3x normal dose
- All patients should wear a Medical Alert bracelet
- All patients should be prescribed and taught how to use IM hydrocortisone

November 2023

Morgan Lapierre (Medical Student, University of Alberta) and Dr. Elizabeth Rosolowsky (Pediatric Endocrinologist, University of Alberta) for www.pedscases.com

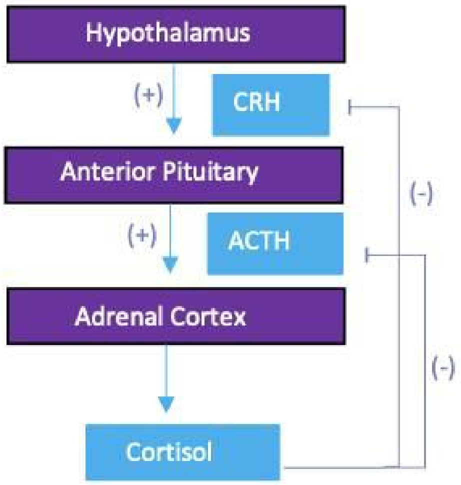
ADRENAL CRISIS

What is an Adrenal Crisis?

- Acute, life-threatening presentation of adrenal insufficiency
- Requires urgent medical intervention to prevent progression to coma and death

Adrenal Insufficiency:

- Inadequate synthesis of glucocorticoids (cortisol) +/- mineralocorticoids (aldosterone) depending on the etiology.



CLINICAL PRESENTATION

ADRENAL INSUFFICIENCY

- Fatigue
- Lethargy
- Nausea
- Vomiting
- Abdominal Pain
- Weight loss
- Poor Growth
- Salt Cravings (Primary AI)
- Hyperpigmentation (Primary AI)

ADRENAL CRISIS

- Dehydration
- Dizziness
- Syncope
- Altered Mental Status
- Seizures
- Hypotension
- Tachycardia
- Coma
- Hypoglycemia

COMMON ETIOLOGIES

ADRENAL INSUFFICIENCY

Primary:

- Decreased cortisol production in the adrenal gland
- Examples: Addison's Disease, Congenital Adrenal Hyperplasia (CAH)

Secondary/Tertiary:

- Decreased ACTH or CRH secretion (leading to low cortisol)
- Examples: acquired/congenital hypopituitarism, excessive glucocorticoid use

ADRENAL CRISIS

- First presentation of a patient with Adrenal Insufficiency (AI)
- Insufficient Glucocorticoid replacement in patient with known AI
- Inadequate Stress Dosing in a patient with known AI in the context of stressors such as: surgery, physical trauma, infection
- Abrupt discontinuation of exogenous steroids

MANAGEMENT

ACUTE

- IV Isotonic Fluid bolus, 20ml/kg
- IV Hydrocortisone 50-100 mg/m2 bolus followed by 50-100 mg/m2/day div q6h
- Dextrose bolus if hypoglycemic
- Treat underlying cause if crisis is triggered by infection or trauma

PREVENTATIVE

- Maintenance hydrocortisone +/- mineralocorticoid replacement
- Stress Dose teaching to patient/families
- Medical Alert Bracelet
- IM Hydrocortisone injection for emergencies
- Detailed emergency action plan
- Monitor for ongoing electrolyte disturbances

WHAT IS STRESS DOSING?

- In a patient with AI, the body requires extra cortisol during times of stress/illness
- Cortisol replacement doses are increased temporarily during this time period
- Common indications: fever, vomiting, diarrhea, fractures

March 2024
Surina Grover (Medical Student, University of Alberta) and Dr. Elizabeth Rosolowsky (Pediatric Endocrinologist, Stollery Children's Hospital) for www.pedscases.com

APPROACH TO ANEMIA

Anemia is a lab finding of reduced hemoglobin concentration, to <2.5%ile for age and sex

Etiology can be broadly classified into three categories:

1. Decreased production of RBC
2. Destruction of RBC
3. Loss/sequestration of RBC

PRESENTATION

History

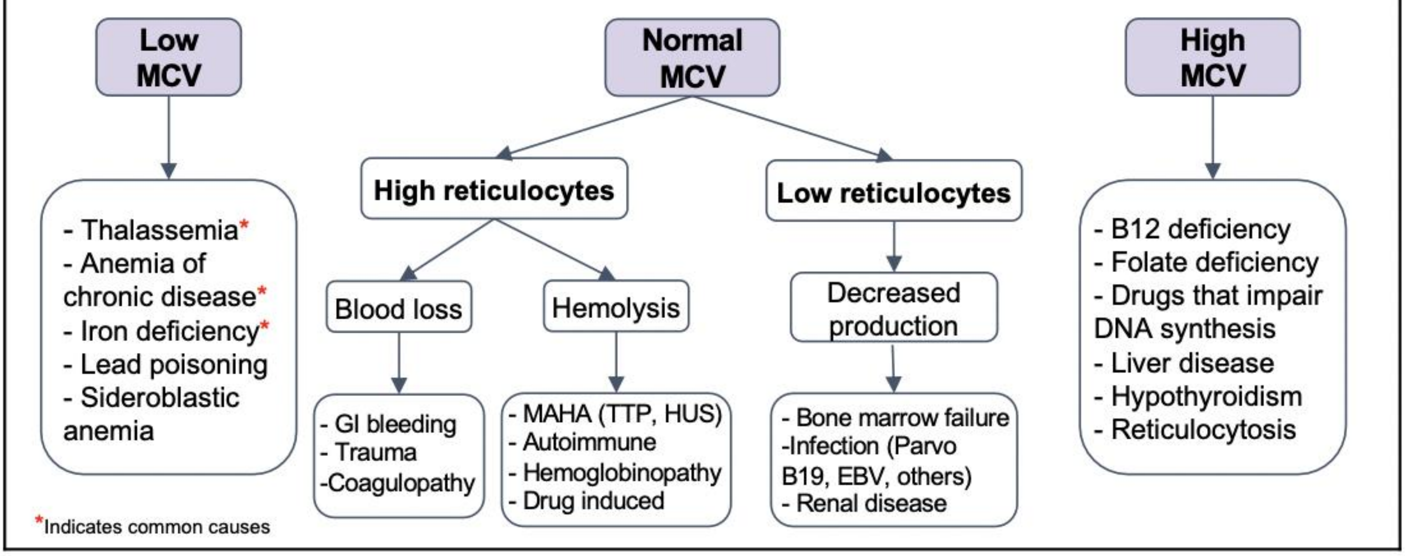
- Fatigue and lethargy
- Exercise intolerance, weakness
- Changes in school performance
- Bleeding (GI, dark urine, menstrual)

Red flag symptoms
Decreased LOC, change in urine colour, jaundice, multiple cell lines affected

PHYSICAL EXAM

- Tachycardia
- Pallor: mucous membranes, conjunctiva, nails, palms
- Fatigue
- Petechiae, purpura
- Jaundice
- Scleral icterus
- Early systolic flow murmur
- Splenomegaly
- Hepatomegaly

ETIOLOGY BASED ON MCV



Low MCV
- Thalassemia*
- Anemia of chronic disease*
- Iron deficiency*
- Lead poisoning
- Sideroblastic anemia

Normal MCV

High reticulocytes

Blood loss
- GI bleeding
- Trauma
- Coagulopathy

Hemolysis
- MAHA (TTP, HUS)
- Autoimmune
- Hemoglobinopathy
- Drug induced

Low reticulocytes

Decreased production
- Bone marrow failure
- Infection (Parvo B19, EBV, others)
- Renal disease

High MCV
- B12 deficiency
- Folate deficiency
- Drugs that impair DNA synthesis
- Liver disease
- Hypothyroidism
- Reticulocytosis

*Indicates common causes

INVESTIGATIONS

Initial labs:

- CBC differential→ to rule out other cytopenias
- Reticulocytes
- +/- Peripheral blood smear

Confirmatory testing, based on MCV/ reticulocytes and history

- if low MCV & hx requires further work-up: ferritin (+/- iron studies), hemoglobinopathy workup
- If considering hemolysis:
  - LDH, bilirubin, haptoglobin, DAT, smear
- if high MCV: liver enzymes TSH, B12, folate
- Other testing based on history/exam

MANAGEMENT

A) Hemodynamically stable

- Treat underlying cause

B) Hemodynamically unstable or severely symptomatic

- Consider transfusion - no specific Hgb cut off value, decision is made based on **acuity, etiology, signs/symptoms**

Iron Deficiency Anemia*

- 1st line: increase dietary iron intake + oral iron therapy at 4-6 mg/kg/day of elemental iron
- 2nd line: above + IV iron therapy

November 2023
Ioulia Opotchanova (Medical Student, University of Alberta), Dr. Jessica Foulds, (Pediatric Hospitalist, University of Alberta) for www.pedscases.com

APPROACH TO ACUTE OTITIS MEDIA (AOM)

Acute otitis media is an infection of the middle ear!

DIAGNOSTIC CRITERIA

1. Middle ear effusion and inflammation
2. Acute onset of symptoms (<48 hours)

COMMON PRESENTATIONS

- Ear Pain
- Ear Fullness
- Fever
- Otorrhea
- Hearing Loss

Children can also appear fussier!

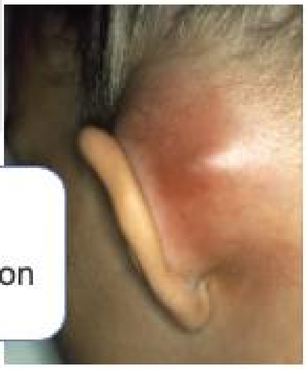
RISK FACTORS

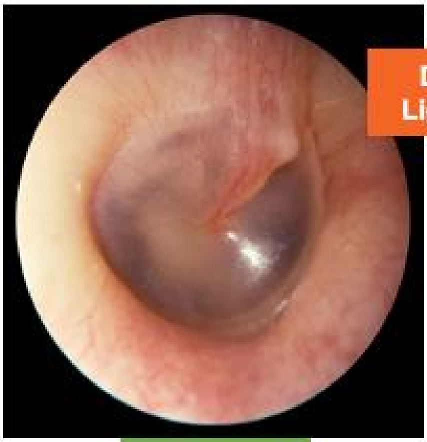
Recent URTI	Sick Contacts (daycare, crowding)
Previous Episodes of AOM	Immuno-deficiency
GERD	Sibling with AOM
Ethnicity (Indigenous)	Cleft Palate/ Lip
Exposure to Cigarette Smoke	Exposure to Air Pollution
Lack of Breastfeeding	Pacifier Use

PHYSICAL EXAM

- Check for fever!
- Inspect and palpate the auricle, peri-auricular region and mastoid process (erythema, swelling, tenderness)
- Look inside the ear with an otoscope!

Remember that mastoiditis is a common complication of AOM!





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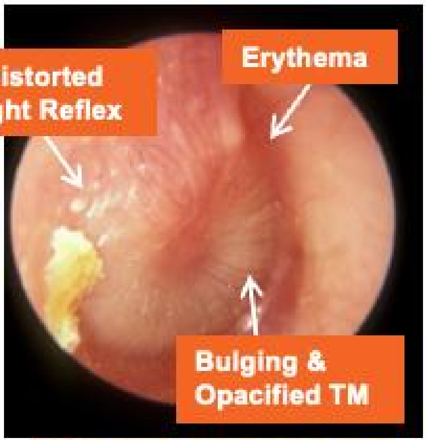

Distorted Light Reflex
Erythema
Bulging & Opacified TM

Acute Otitis Media

Published December 2020
Joanna Yuen, Sahar Saleem (Medical Students 2021, University of Alberta), Michael Prodanuk (PGY3, University of Toronto), Dr Joan Robinson for www.pedscases.com

APPROACH TO ACUTE OTITIS MEDIA (AOM)

GENERAL MANAGEMENT

1. Treat pain and fever with analgesics (ibuprofen, acetaminophen)
2. Assess whether the patient requires antibiotics

Do all cases of AOM need antibiotics?

AGE OF CHILD

≤ 6 months old

Start Antibiotics

> 6 months old

Assess Clinical Status

Mildly bulging TM, Mild symptoms and Temp <39°C

Observe for 48 hours

Symptoms not improving or complications (hearing loss, gait imbalance, mastoiditis, perforated TM)

Start Antibiotics

Perforated tympanic membrane with purulent discharge or bulging tympanic membrane with one of the following:

- **Fever (temp ≥ 39°C)**
- **Moderately or severely ill (irritability, severe otalgia, difficulty sleeping)**
- **Symptoms lasting > 48 hr**

Start Antibiotics

1st Line Antibiotic: AMOXICILLIN
Dose: 90 mg/kg/day PO divided BID-TID
Duration: 5 days if ≥ 2 y/o; 10 days if < 2 y/o, perforated TM, or recurrent AOM

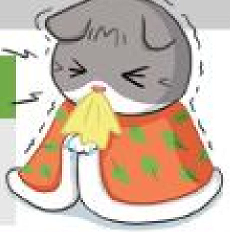
The most common bacterial causes of AOM are Strep Pneumonia, H influenza and M Catarrhalis!

COMPLICATIONS

Mastoiditis, effusion, hearing loss, learning difficulties, meningitis, gait imbalance

Alternative Antibiotics:

- **Amoxicillin-Clavulanate** (if amoxicillin used in the last 30 days or treatment failure with amoxicillin)
- **Cefuroxime-axetil or Ceftriaxone** (for non-anaphylaxis penicillin allergy)
- **Clarithromycin or Azithromycin or Clindamycin** (for anaphylaxis penicillin allergy)
- **Ceftriaxone** (if failure of amox-clav or oral drugs not tolerated or persistent symptoms while on antibiotics >3 days)



PREVENTION

- Decrease modifiable risk factors (exposure to cigarette smoke, bottle feeding)
- Encourage hand hygiene, Pneumo-13 vaccination, annual influenza vaccine

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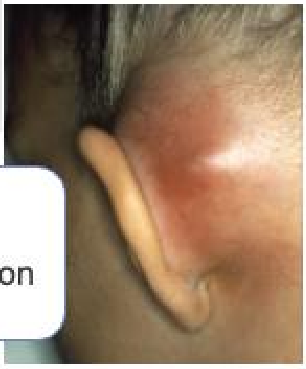

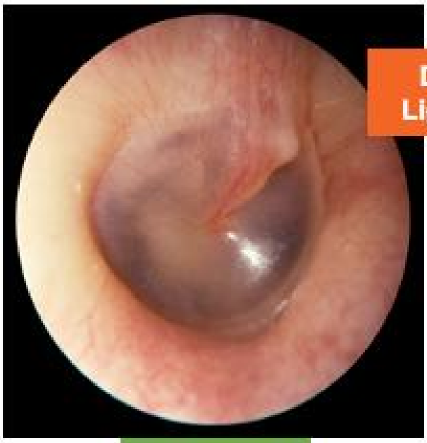

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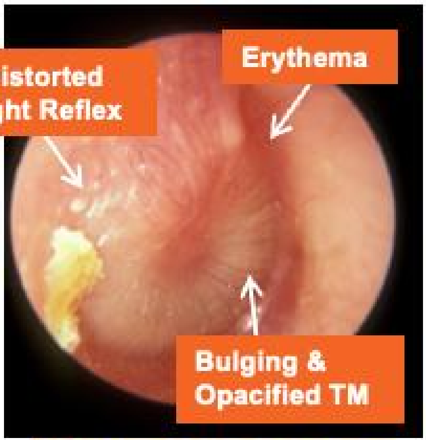

Acute Otitis Media

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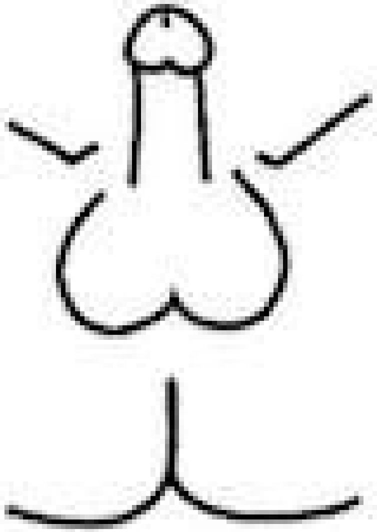

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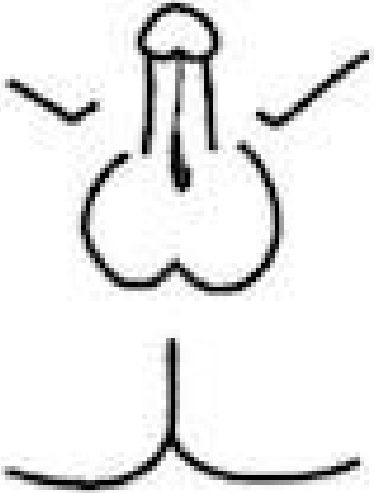

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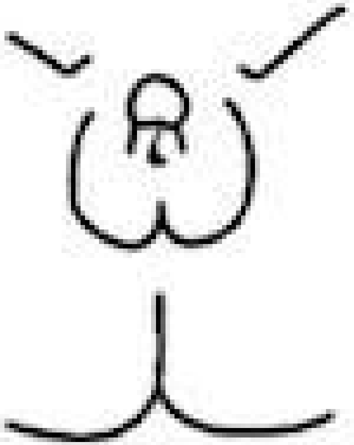

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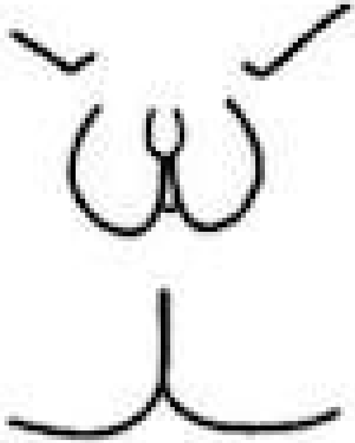

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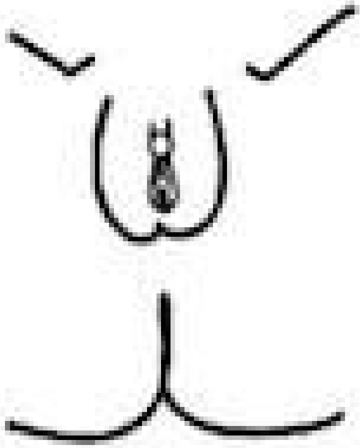

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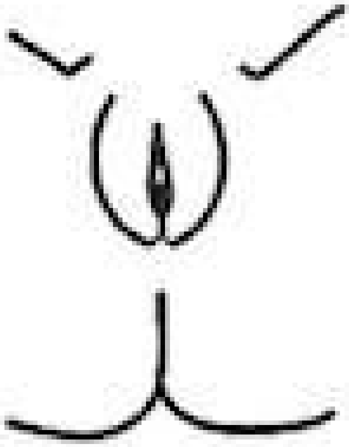

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APPROACH TO ANEMIA

Anemia is a lab finding of reduced hemoglobin concentration, to <2.5%ile for age and sex

Etiology can be broadly classified into three categories:

1. Decreased production of RBC
2. Destruction of RBC
3. Loss/sequestration of RBC

PRESENTATION

History

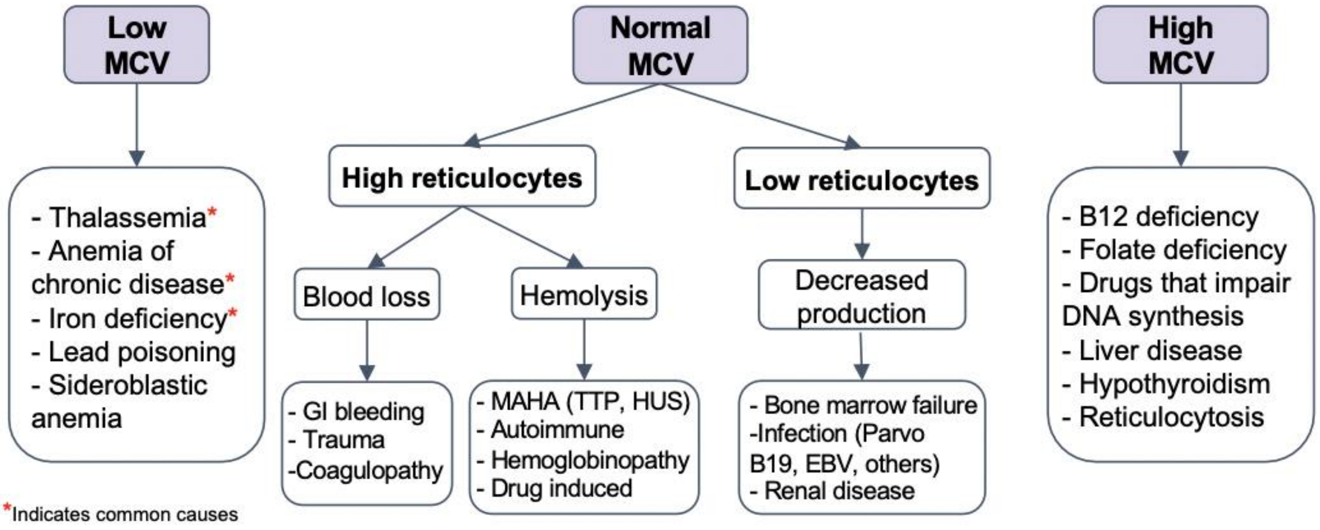
- Fatigue and lethargy
- Exercise intolerance, weakness
- Changes in school performance
- Bleeding (GI, dark urine, menstrual)

Red flag symptoms
Decreased LOC, change in urine colour, jaundice, multiple cell lines affected

PHYSICAL EXAM

- Tachycardia
- Pallor: mucous membranes, conjunctiva, nails, palms
- Fatigue
- Petechiae, purpura
- Jaundice
- Scleral icterus
- Early systolic flow murmur
- Splenomegaly
- Hepatomegaly

ETIOLOGY BASED ON MCV



Low MCV

- Thalassemia*
- Anemia of chronic disease*
- Iron deficiency*
- Lead poisoning
- Sideroblastic anemia

Normal MCV

High reticulocytes

Blood loss
- GI bleeding
- Trauma
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Hemolysis
- MAHA (TTP, HUS)
- Autoimmune
- Hemoglobinopathy
- Drug induced

Low reticulocytes

Decreased production
- Bone marrow failure
- Infection (Parvo B19, EBV, others)
- Renal disease

High MCV

- B12 deficiency
- Folate deficiency
- Drugs that impair DNA synthesis
- Liver disease
- Hypothyroidism
- Reticulocytosis

*Indicates common causes

INVESTIGATIONS

Initial labs:

- CBC differential→ to rule out other cytopenias
- Reticulocytes
- +/- Peripheral blood smear

Confirmatory testing, based on MCV/ reticulocytes and history

- if low MCV & hx requires further work-up: ferritin (+/- iron studies), hemoglobinopathy workup
- If considering hemolysis:
  - LDH, bilirubin, haptoglobin, DAT, smear
- if high MCV: liver enzymes TSH, B12, folate
- Other testing based on history/exam

MANAGEMENT

A) Hemodynamically stable

- Treat underlying cause

B) Hemodynamically unstable or severely symptomatic

- Consider transfusion - no specific Hgb cut off value, decision is made based on **acuity, etiology, signs/symptoms**

Iron Deficiency Anemia*

- 1st line: increase dietary iron intake + oral iron therapy at 4-6 mg/kg/day of elemental iron
- 2nd line: above + IV iron therapy

November 2023

Ioulia Opotchanova (Medical Student, University of Alberta), Dr. Jessica Foulds, (Pediatric Hospitalist, University of Alberta) for www.pedscases.com

pedscases notes

APPROACH TO BRONCHIOLITIS

Pathophysiology

- Viral lower respiratory tract infection
- Obstruction of small airways due to inflammation and mucus production
- **Etiology**: Respiratory syncytial virus (RSV), human metapneumovirus, influenza, rhinovirus, adenovirus, and parainfluenza

Presentation – Symptoms peak at day 3-5 of illness, then improve

Wheezing
Cough

Respiratory distress
Apnea

Rhinorrhea
Nasal congestion

Dehydration

Fever

Bronchiolitis	versus	Asthma
First episode wheeze (but can recur)		Recurrent wheeze
Typically <1 year (can be up to 2 years)		Typically >1 year
No bronchospasm = No response to salbutamol		Bronchospasm = Response to salbutamol

Investigations – None required. Clinical diagnosis based on signs and symptoms!

- **Chest X-ray**
  If suspecting alternate diagnosis
- **Blood Gas**
  If concerned about respiratory failure
- **Respiratory Virus Swab**
  For hospital cohorting or suspected influenza (not routinely recommended)
- **Other Blood Work or Cultures**
  For severely ill or febrile infants <3 months old

Management

Indications for Admission: Requirement of supplemental oxygen, dehydration, apnea, risk factors for severe disease, severe respiratory distress

Recommended:

Oxygen
Maintain saturation >90%

Hydration
Encourage oral feeds.
Consider NG tube or IV fluids

Severe: Consider high-flow oxygen, non-invasive ventilation, and intubation

Equivocal Evidence:

- Nebulized epinephrine
- Nebulized hypertonic saline
- Nasal suctioning

NOT Recommended:

- Salbutamol
- Glucocorticoids
- Antibiotics
- Antivirals
- Chest physiotherapy
- Aerosolized saline

May 2020
Viane Faily (Medical Student, University of Alberta) for www.pedscases.com

APPROACH TO HEART MURMURS

History

- FTT, FHx
- Dyspnea, palpitations
- Exercise intolerance
- Dizziness, syncope
- Chest pain on exertion

Physical Exam

- Vital signs
- Cyanosis
- Clubbing
- Weak/absent femoral pulses
- Pulses and capillary refill
- ↑ precordial activity
- Dysmorphic features
- Hepatomegaly

ANY 🚩 on history or physical exam makes it a **pathologic** murmur! (regardless of auscultation)

Auscultation

Innocent Physiologic, no heart disease		**Pathologic** Underlying heart disease
Systolic	timing	Diastolic, holosystolic
Soft/vibratory	quality	Harsh
Grade II or less	intensity	Grade III or higher (possible thrill)
Exercise, anemia, fever	louder with	Usually NO change with position
NO extra sounds	other sounds	Click or opening snap, S3, S4
Normal S2 (physiologic split on inspiration)		Fixed split S2

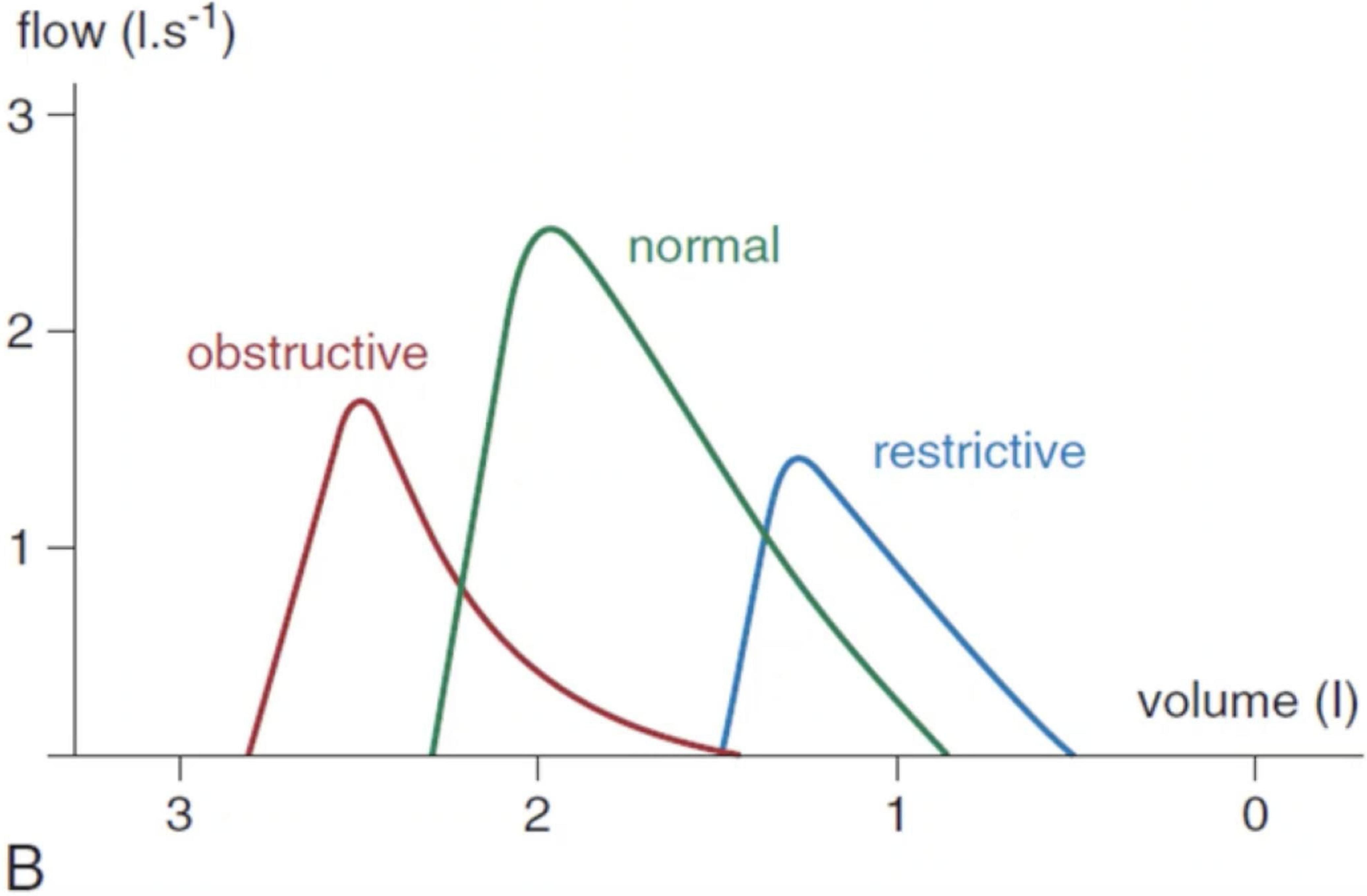
Must have **ALL** features to be an **innocent** murmur

ANY pathologic feature makes it a **pathologic** murmur

Refer to a pediatric cardiologist when there is…

- Suspected **pathologic** cause or lingering **uncertainty**
- FHx of **congenital heart disease** in 1st degree relative
- FHx of **Marfan** syndrome or **unexplained/sudden cardiac death** in young person
- Known/suspected **chromosomal/genetic** conditions (ex. Trisomy 21)
- **Request** from parents within good reason

November 2019
Sarah Park (MD Student 2022, University of Alberta) for www.pedscases.com


flow (l.s⁻¹)
3
2
1
normal
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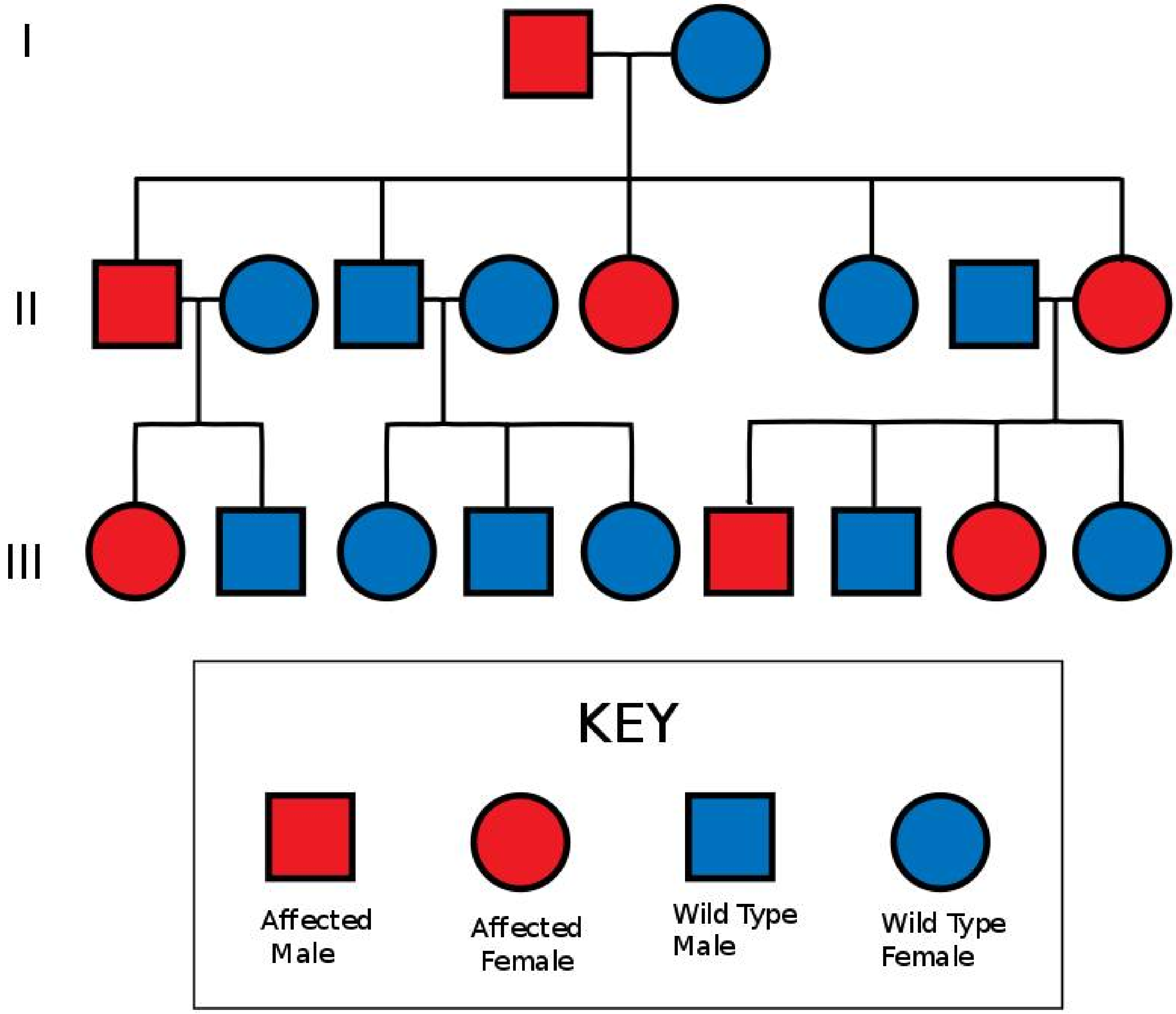

I
II
III
KEY
Affected Male
Affected Female
Wild Type Male
Wild Type Female

APPROACH TO THERMAL BURNS

KEY POINTS

Burns are a leading cause of unintentional injury and death in children

1. Burn injuries should be managed as a trauma case and require primary and secondary survey
2. Total Body Surface Area (TBSA) calculation is essential for adequate fluid resuscitation

BURN ASSESSMENT

- A burn diagram, (the pediatric Lund and Browder chart), is used to estimate the total body surface area (TBSA) of the burn (represented as a % of body surface area)

Superficial burns** are **not included** in the **TBSA calculation

BURN CENTER REFERALL

1. ≥ 10% TBSA partial and/or full thickness age ≤10
2. Full thickness burns ≥ 5% TBSA any age
3. Inhalation injury
4. Electrical burns (including lightning injury)
5. Concomitant trauma in which the burn injury poses the greatest risk of morbidity and mortality
6. Chemical burns
7. Burned children in hospitals without qualified personnel or equipment for the care of children

***This list is not comprehensive.** Refer to American Burn Association Burn Center Referral Criteria*

PAIN MANAGEMENT

- Mild pain may be managed with acetaminophen and/ or ibuprofen
- Severe pain will require IV opioids as needed (e.g. IV morphine or fentanyl)

EMERGENCY DEPARTMENT ASSESSMENT

- **AIRWAY:** Signs of airway burn/ inhalation injury: stridor, hoarseness, respiratory distress
- **BREATHING:** Full thickness and/or circumferential chest burns may require escharotomy to permit chest expansion
- **CIRCULATION:** IV fluid resuscitation (refer to fluid management)
- **DISABILITY:** If altered conscious state, consider airway support
- **EXPOSURE**: Expose whole body for assessment of burn depth and total body surface area of burn

HISTORY: Mechanism of injury (consider the potential of non-accidental injury and report appropriately if concerns)

CLASSIFICATION OF BURNS BY DEPTH

Burn Depth	Appearance	Sensation	Healing time
Superficial (epidermis only)	Dry, red	Painful	3 to 6 days
Superficial partial-thickness (epidermis and papillary dermis)	Blisters, moist, red, weeping	Painful to temperature, air and touch	7 to 21 days
Deep partial-thickness (epidermis and reticular dermis)	Blisters; Wet or waxy dry; Variable color (patchy white to red)	Painful to pressure only	>21 days, usually requires surgical treatment (skin grafting)
Full thickness (epidermis and dermis)	Waxy white to leathery gray to charred and black; Dry and inelastic	Painful to deep pressure only	Rare, unless surgically treated (skin grafting)

FLUID MANAGEMENT

- For patients with burns measuring greater than 10% TBSA, a burn resuscitation formula such as the Parkland Formula is used to estimate the fluid volume requirements for the first 24 hours

Parkland formula= 4mL x % TBSA burned x Weight (kg)
Give ½ in **first 8h** since the time of injury
Give ½ in the **next 16h**

- Children <30kg should receive maintenance fluid with D5W ½ NS in addition to calculated requirements (use 4:2:1 rule)
- Warmed ringer's lactate is used in fluid resuscitation to prevent hypothermia

September 2023
Emma Yanko (Medical Student, University of Saskatchewan), Dr. Craig Gendron (Pediatric Plastic Surgeon, University of Saskatchewan) for www.pedscases.com

APPROACH TO THROMBOCYTOPENIA

PRESENTATION

HISTORY

Bleeding Hx

- Provoked (trauma, surgery) or spontaneous
- Deep or superficial bleeds

Red Flag Symptoms

! Fever, weight loss, night sweats, bone pain, fatigue

- Prodromal illness
- Recent live vaccination (MMR)
- Recent travel
- Dietary History
- Family Hx of bleeding disorders
- Review Medications list
- If newborn ask about maternal PLT count, medical history and medications

PHYSICAL EXAM

- ABCs and vital signs (stable or unstable)
- Purpura, petechiae, ecchymoses, mucocutaneous bleeding
- Altered consciousness, abnormal pupils, slurred speech
  Lymphadenopathy (malignancy)
- Hepatosplenomegaly
- Swollen joints or rash

INVESTIGATIONS

CBC (isolated PLT vs. pancytopenia)

- Check previous PLT counts

Peripheral blood smear

Bone marrow examination ONLY IF:

! Red Flag Symptoms

! Involvement of other blood cell lines (anemia or neutropenia)

! Blasts on smear

Definition of thrombocytopenia:
platelet (PLT) count < 150,000/μL

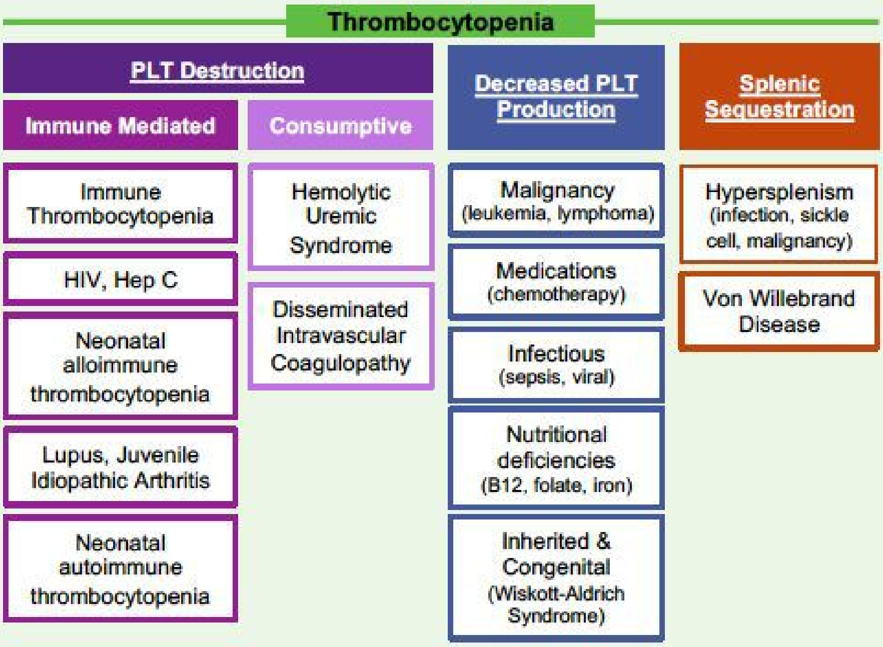
SIGNS AND SYMPTOMS

Platelet (x 10^3 /μL)	Example of Bleeding Risk
> 100	Asymptomatic
50 - 100	Post-operative bleeding & bruising
20 - 50	Petechiae, purpura, ecchymoses
5 - 20	Epistaxis or gingival bleeding
< 10	GI bleeding, heavy menstrual bleeding or intracranial hemorrhage

IMMUNE THROMBOCYTOPENIA (ITP)

- **#1 etiology** in children
- Sudden severe drop in PLT count often associated with petechiae and mild bruising
- Ask about **recent viral illnesses** or **live vaccine** administration
- Majority recover with **observation**, however, may require **corticosteroids** or **IVIG**

DIFFERENTIAL DIAGNOSIS



Thrombocytopenia

- **PLT Destruction**
  - **Immune Mediated**
    - Immune Thrombocytopenia
    - HIV, Hep C
    - Neonatal alloimmune thrombocytopenia
    - Lupus, Juvenile Idiopathic Arthritis
    - Neonatal autoimmune thrombocytopenia
  - **Consumptive**
    - Hemolytic Uremic Syndrome
    - Disseminated Intravascular Coagulopathy
- **Decreased PLT Production**
  - Malignancy (leukemia, lymphoma)
  - Medications (chemotherapy)
  - Infectious (sepsis, viral)
  - Nutritional deficiencies (B12, folate, iron)
  - Inherited & Congenital (Wiskott-Aldrich Syndrome)
- **Splenic Sequestration**
  - Hypersplenism (infection, sickle cell, malignancy)
  - Von Willebrand Disease

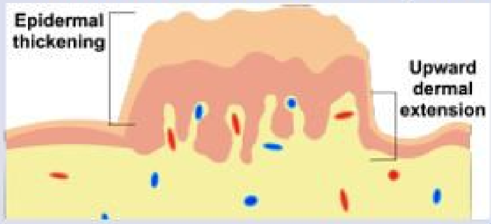
⚠ DISSEMINATED INTRAVASCULAR COAGULOPATHY (DIC) ⚠

- ↑ thrombosis & ↑ bleeding
- Prolonged PTT, INR
- Low fibrinogen
- Sick patient requiring PICU
- Identify and treat the underlying cause (e.g., sepsis, trauma or malignancy)
- PLT transfusion may be indicated if significant bleeding
- Transfusion thresholds vary based on clinical situation (<10-100 x 10^3/μL PLT)
- 5-10 mL/kg PLT transfusion (max 300 mL) over 1 hour

Published August 2021
Usman Ahmed (MD 2022, University of Alberta) & Dr. Mark Belletrutti (Pediatric Hematology and Clinical Associate Professor, University of British Columbia) for www.pedscases.com

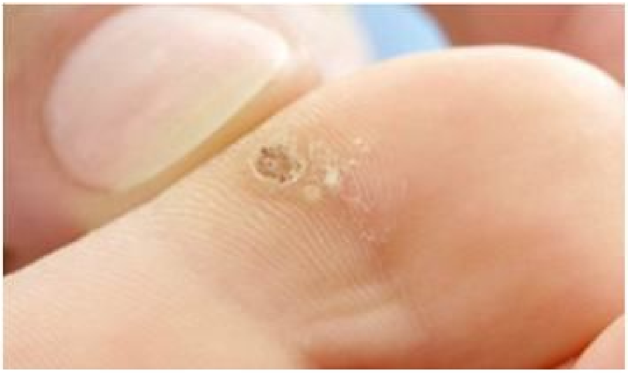
APPROACH TO WARTS (VERRUCA)

- Caused by a Human Papilloma Virus (HPV) infection in epidermal cells, leading to thickening of the epidermis and propagation of underlying dermal layer (with capillaries)
- Common in both healthy children and immunosuppressed patients
- Anogenital warts should raise suspicion of sexual abuse, although other transmission modes are possible



DIAGNOSIS

- ☐ Diagnosed by clinical appearance of thrombosed capillaries under a hyperkeratotic cap
- ☐ Differential Diagnosis → callus, closed comedone, lichen planus, corn, molluscum contagiosum



MANAGEMENT

Common/Plantar Warts (*Verruca vulgaris*)

At home:

- First line treatment
- Apply 17% salicylic acid and cover with duct tape
- Repeat daily for 12wks

In office cryotherapy:

- Cryotherapy is a good option in children, but may require topical anesthetic to be able to tolerate treatment
- Treat, wait for thaw, and repeat
- Follow up in 4wks

Flat Warts

- Topical tretinoin 0.1% cream (off-label) applied daily; follow up in 4wks

Anogenital Warts (*Condyloma acuminatum*)

At home:

- Imiquimod cream (off-label for children); use with varied frequency depending on the strength
- Sinecatechins (ex Veregen) ointment (off-label); apply a thin layer TID for up to 16wks; do not use in the vagina/anus

Note:

Alternative treatments may include laser ablation or intralesional bleomycin

Course & Complications

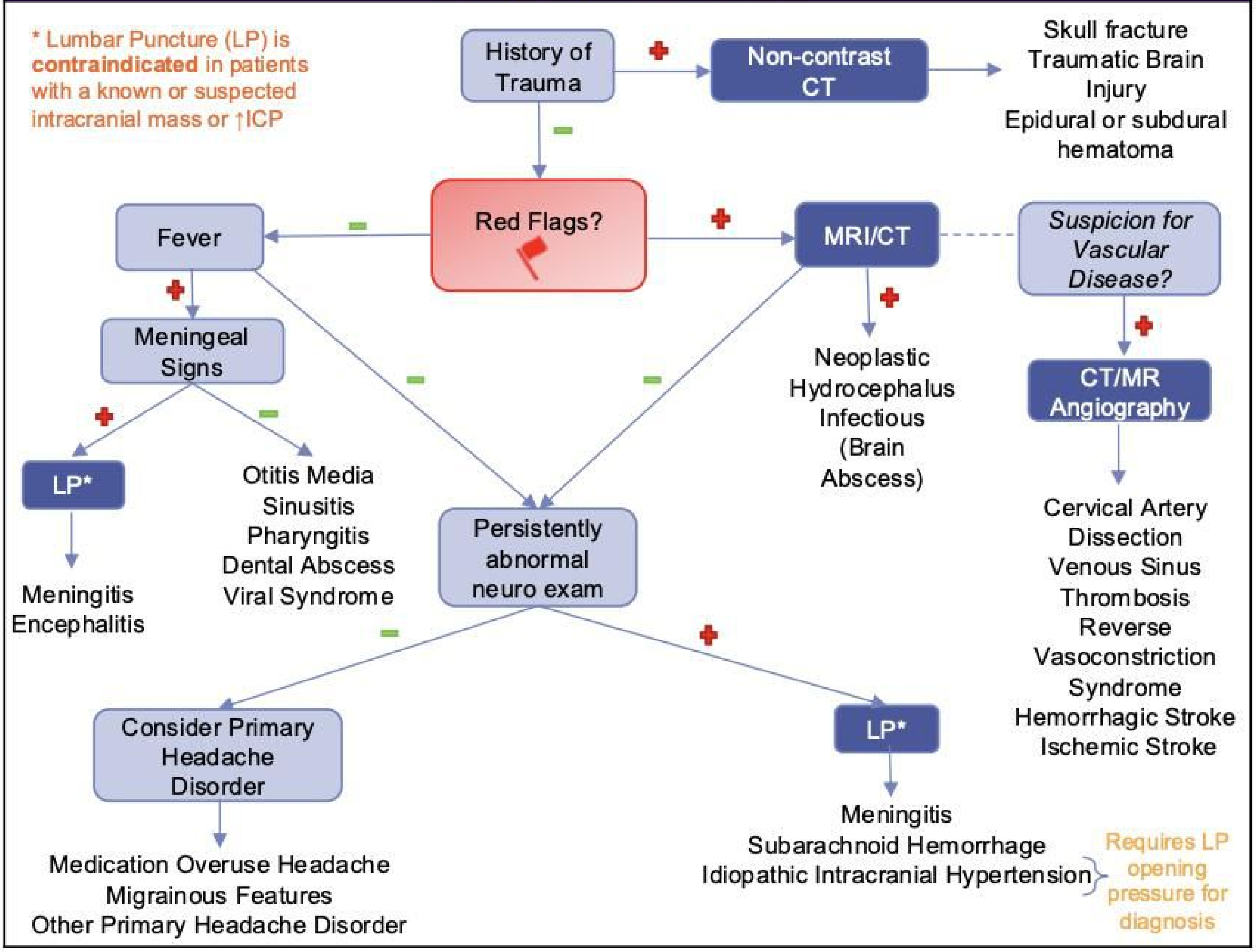
- Up to 65% of patients achieve spontaneous remission within 2 years, thus watching and waiting a reasonable treatment
- Blister-forming agents may result in new wart formation at periphery
- Patients with more pigmented skin may get dyspigmentation from treatment options
- Consider referral to a dermatologist for complicated warts or warts refractory to treatment

Published May 2021

Colton Jensen (Medical Student 2023, University of Alberta) and Dr. Miriam Weinstein (Pediatrician and Dermatologist, Hospital for Sick Children) for www.pedscases.com

ASSESSMENT OF HEADACHE IN THE EMERGENCY ROOM

ACUTE SEVERE HEADACHE



* Lumbar Puncture (LP) is **contraindicated** in patients with a known or suspected intracranial mass or ↑ICP

History of Trauma
+ → Non-contrast CT → Skull fracture, Traumatic Brain Injury, Epidural or subdural hematoma
− → Red Flags?

Red Flags?
+ → MRI/CT
− → Fever

MRI/CT
+ → Neoplastic, Hydrocephalus, Infectious (Brain Abscess)
− → Persistently abnormal neuro exam

MRI/CT - - - *Suspicion for Vascular Disease?*
+ → CT/MR Angiography → Cervical Artery Dissection, Venous Sinus Thrombosis, Reverse Vasoconstriction Syndrome, Hemorrhagic Stroke, Ischemic Stroke

Fever
+ → Meningeal Signs
− → Persistently abnormal neuro exam

Meningeal Signs
+ → LP* → Meningitis, Encephalitis
− → Otitis Media, Sinusitis, Pharyngitis, Dental Abscess, Viral Syndrome

Persistently abnormal neuro exam
− → Consider Primary Headache Disorder → Medication Overuse Headache, Migrainous Features, Other Primary Headache Disorder
+ → LP* → Meningitis, Subarachnoid Hemorrhage, Idiopathic Intracranial Hypertension

Idiopathic Intracranial Hypertension } Requires LP opening pressure for diagnosis

HISTORY

- Abrupt onset (“worst headache of my life”, thunderclap)
- Positional association
- Occurs soon after waking or awakens from sleep
- Precipitated by Valsalva
- Progressive and worsening over time
- Focal or global neurological deficits

PHYSICAL EXAM

- Hypertension with brady- or tachycardia → suggestive of increased intracranial pressure (ICP)
- Prolonged altered mental status (>60 minutes)
- Papilledema
- Neck stiffness
- Visual Changes
- Ataxia
- Dysmetria
- Focal neurologic deficits (e.g. cranial nerve palsy, motor, somatosensory)

INDICATIONS FOR NEUROIMAGING

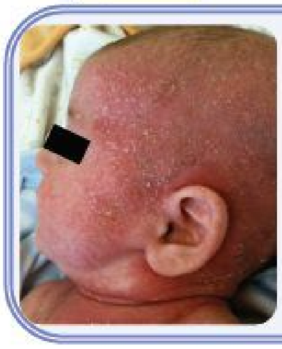
Red flag findings: Focal neuro deficits, concern for ↑ICP, thunderclap onset
- Non-contrast CT – rapid assessment
- CT – often first-line if clinically stable
- Inpatient MRI
- Consider Angiography (CTA or MRA) if suspicious for vascular etiologies

Chronic, progressive headache with normal neuro exam:
- Expedited outpatient MRI

Neurocutaneous findings:
- Non-urgent MRI
- Genetic workup

November 2024
Julia Craig (Medical Student, University of Alberta), Dr. Thilinie Rajapakse (Pediatric Neurologist, University of Alberta) for www.pedscases.com

ATOPIC DERMATITIS (AD)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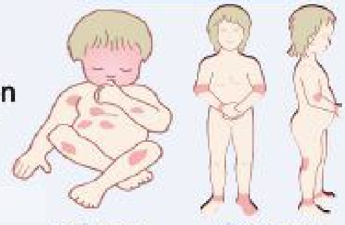
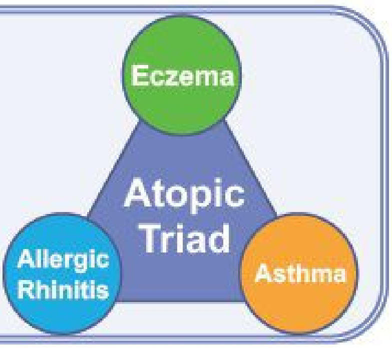
Atopic dermatitis (also known as eczema) is a **common chronic inflammatory** skin condition that begins during **infancy or early childhood**, characterized by **dry, pruritic, erythematous** papules and plaques

Pattern of AD



Infants < 4 yo | Children > 4 yo



Atopic Triad: Eczema, Allergic Rhinitis, Asthma

CLINICAL FEATURES

Diagnostic criteria: itchy skin (essential) + ≥ 3 of the following:

1. Hx of relapsing **OR** current visible **flexural dermatitis (> 4 yo)** or dermatitis on **cheeks, forehead, outer limbs (< 4 yo)**
   See pattern of AD above
2. Personal Hx of **asthma** or **allergic rhinitis**(or positive FHx of first-degree relative in children < 4 yo)
3. **Xerosis** within past year
4. Onset < 2 years

PATHOPHYSIOLOGY

Epidermal Barrier Dysfunction
Genetic Disposition
Immune Dysregulation

QUALITY OF LIFE

- Psychosocial distress (loss of sleep, intense pruritis, social embarrassment)
- ↑ risk of ADHD, depression, and anxiety

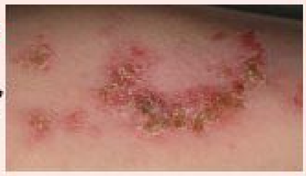
DIFFERENTIAL DIAGNOSIS

- **Seborrheic / contact / nummular dermatitis**
- **Infestations -** scabies
- **Infection -** tinea, impetigo
- **Autoimmune -** lupus, dermatomyositis
- **Primary immunodeficiencies** (rare)
- **Mycosis fungoides** (in adolescents)

COMPLICATIONS

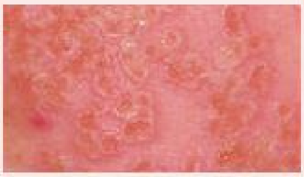
IMPETIGO

- *S. aureus* infection
- Yellow overlying "honey-crusting"
- Topical/systemic antibiotics



ECZEMA HERPETICUM

- HSV infection
- Monomorphic, punched out erosions
- Immediate antiviral therapy



ECZEMA COXSACKIUM

- Coxsackie virus infection

MANAGEMENT

NON-PHARMALOGICAL (All patients)

- **Patient education** (www.eczemahelp.ca)
- **Eliminate exacerbating factors** (e.g. scented products, dry air)
- Skin **hydration** (e.g. petroleum jelly, thick unscented creams)

MILD TO MODERATE DISEASE

- **Topical corticosteroids** (BID)
  - Face: Hydrocortisone 1% ointment
  - Body: Betamethasone valerate 0.05% ointment
- Topical calcineurin inhibitors
- Topical phosphodiesterase-4 inhibitor

MODERATE TO SEVERE DISEASE

- **Phototherapy**
- **Immunosuppressants**
- **Biologics** (dupilumab)

Published July 2021
Selena Osman (MD Student 2023, University of Calgary), Dr. Harry Liu (Dermatology Resident, UBC), and Dr. Michele Ramien (Pediatric Dermatologist, University of Calgary) for www.pedscases.com

BECKWITH-WIEDEMANN SYNDROME

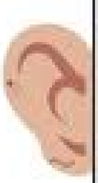
An **overgrowth disorder** resulting from epigenetic or genetic alterations, such as **abnormal methylation** or a pathogenic variant in CDKN1C (Cyclin-Dependent Kinase Inhibitor 1C). It follows an **autosomal dominant** inheritance pattern, though most cases are **sporadic**.

AD
Autosomal dominant

CLINICAL FEATURES

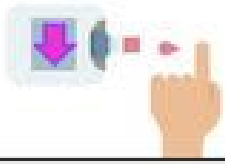
Growth:
- **Macrosomia**
- **Hemihypertrophy**
- Rapid growth
- Growth rate slows after 8 years of age

Facial features:
- **Macroglossia**
- **Earlobe creases**
- Earlobe pits
- Nevus flammeus
- Infraorbital creases



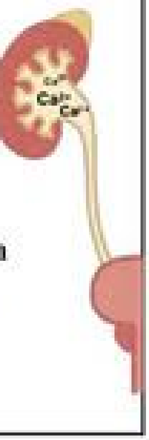
Gastrointestinal:
- **Omphalocele**
- **Umbilical hernia**
- Visceromegaly

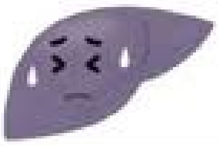
Endocrine:
- **Hypoglycemia**
- Persistent hyperinsulinism



Renal:
- Medullary dysplasia
- **Nephrocalcinosis**

Embryonal tumors:
- **Wilms tumor**
- **Hepatoblastoma**
- Neuroblastoma
- Rhabdomyosarcoma





DIAGNOSIS

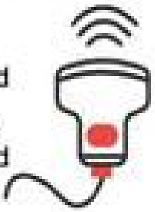
- Identification of **key clinical features**

Confirmatory Genetic Testing:
- **DNA methylation studies**
- **Chromosomal microarray** to confirm microdeletions/ duplications, or paternal uniparental disomy

CANCER SCREENING

Early diagnosis and regular cancer screening are essential.

- **Abdominal ultrasound**
  - Every *3 months* until 8 years old
- **Alpha-fetoprotein level (AFP)** levels
  - Every *3 months* until 4 years old
- **Renal bladder ultrasound**
  - *Annually* from 8 years to adolescence



MANAGEMENT

- **Genetic counseling**: to understand genetic risks and implications.
- **Developmental monitoring**: track milestones and address any delays.
- **Macroglossia**: affects ~ 90% of children with BWS.
  - Feeding support and/or surgical management.
- **Abdominal wall defects**: may require surgical repair.
- **Hypoglycemia management**: treatment may involve diazoxide, octreotide, continuous feeds, or partial pancreatectomy.
- **Leg-length differences**: orthotics or other methods can be used to address differences in leg length.

February 2025
Dr. Katharine V. Jensen (Pediatric Resident, University of Alberta) and Dr. Karen Forbes (Professor of Pediatrics, University of Alberta) for www.pedscases.com

BREASTFEEDING & INFANT FORMULAS

Best practice: Breastfeeding exclusively for first 6 months

BENEFITS

- ✓ **Breastmilk**: easily digestible, supports optimal growth, contains immunoglobulins + white blood cells, protects against GI infections, acute otitis media, respiratory tract infection & more
- ✓ Breastfeeding can promote maternal-infant bonding

Contraindications (Rare)

Maternal Medications: Chemotherapy agents, Radioactive isotope drugs
Maternal Medical Conditions: HIV; herpes lesions on both breasts; critical illness, untreated tuberculosis
Infant conditions: galactosemia ⚠

Breastfed infants require **Vitamin D3 400 units PO daily** to prevent rickets

CHALLENGES

- Breastfeeding isn't for everyone, and some mothers may be unable to or choose not to.
- Respect these decisions without judgement and work together to create a plan that keeps babies healthy & growing.
- Mothers can encounter difficulties with latching and can try expressing breast milk by hand or mechanical pump.
- Expressed breast milk (EBM) is nutritionally equivalent to breastfeeding.

Next best: Commercial iron-fortified cow's milk-based formula

Cow's milk based	Soy milk based	Partially Hydrolyzed	Extensively Hydrolyzed Protein	Amino Acid Based (elemental)	Premature formulas
First line	**Indication**: Galactosemia	Proteins are partially broken down (marketed as easier to digest) May reduce diarrhea in acute gastroenteritis	Proteins are fully broken down **Indications**: • Food protein induced allergic proctocolitis (cow's milk/soy protein intolerance) • GERD • Confirmed food allergies, malabsorption syndromes **Cons**: Higher cost, poor palatability	**Indications**: multiple food protein intolerances who cannot tolerate extensively hydrolyzed casein infant formulas **Cons**: Typically higher cost, poor palatability, and limited availability	Designed to help preemies have good catch-up growth Typically have more calories, protein, vitamins and minerals (higher in iron)

Caloric content: 0.68 kcal/mL in breastmilk and standard formulas, 0.80 kcal/mL in premature formulas

- ⓘ Infants should **not** be fed evaporated milk, cow's/goat's milk, or plant milks
- ⓘ Ingesting cow's milk <6 months old can lead to occult blood loss in stool and anemia

POWDERED FORMULA PREPARATION

Always clean surfaces + sterilize all equipment by boiling in a pot of water for 2 minutes. Allow equipment to air-dry

Powdered formula: Not yet sterile. Obtain tap water, bring to a boil for at least 2 minutes to sterilize. Let the water cool to room temperature (below 70ºC). Mix formula and water, following directions on the label. Store in a fridge and use mixed **formula within 24 hours**.

Published June 2021
Michelle Schneeweiss (Medical Student, McMaster University) and Dr. Jason Silverman (Pediatric Gastroenterologist, University of Alberta) for www.pedscases.com

BREASTFEEDING

Physiology Reflexes

Baby Suckles → Anterior Pituitary secretes **Prolactin** → ↑ Milk production → ↑ Suckling

Baby Suckles → Posterior Pituitary secretes **Oxytocin** → **Let Down Reflex** → ↑ Milk release

Nutritional Components

- ✓ 3.5g/100mL of **fatty acids**
- ✓ Lactose and oligosaccharide **CHOs**
- ✓ 0.9g/100mL of **proteins**
- ✓ Breastmilk has enough calories for infants to meet their growth requirements

Breastfed babies require **400-800 IU** of **Vitamin D supplementation** daily!

Benefits to Mother & Baby

- ❖ ↓ risk of **postpartum bleeding**
- ❖ Decrease **stress** for mom & baby
- ❖ Improve **host defense**
- ❖ Promote **GI** development
- ❖ ↑ **bonding** of mom & baby

Common Concerns	Approaches
Insufficient milk supply	✓ Frequent **feeding** every 2-3 hours ✓ Sufficient **milk removal** by feeding or pumping
Poor latch	✓ Try correcting techniques and **repositioning** ✓ Get support from a **lactation consultant**
Nipple and Breast Pain	✓ **Lanolin cream** is helpful for sore or cracked nipples ✓ **Mastitis** should be assessed by an MD
Jaundice	✓ Lactation failure jaundice requires **medical intervention** ✓ Physiologic jaundice is benign and resolves by itself
Unable to Breastfeed	✓ If moms cannot/do not wish to breastfeed, that's ok! ✓ Many options available for **formula feeding** ✓ Provide counselling and **support**

Overall, the most important thing to consider is that the baby is well fed, growing and thriving, and the mother feels well supported!

April 2022
Jennifer Butler (Medical Student, McMaster University) and Dr. Melanie Lewis for www.pedscases.com

BRUE

Brief

The observer reports a **sudden, brief episode (< 1 minute)**

Resolved

The episode is now resolved

Unremarkable physical exam

Unexplained

Diagnosis of exclusion

No explanation after conducting history and physical exam

Event

≥ 1 of the following:

- **Central cyanosis** or **pallor**
- Absent, decreased, or irregular **breathing**
- Change in **tone** (hyper- or hypotonia)
- Altered **level of responsiveness**

< 1 YEAR OLD
Must meet **all the criteria** to diagnose a BRUE

If an **explanation is found**, then it's **NOT a BRUE**

LOW RISK

- Well child
- No red flags
- Recurrence is unlikely

HIGH RISK

- **Red flags present**
- Requires further investigations

RED FLAGS

- Young age (< 60 days old)
- > 1 episode
- Event lasting > 1 minute
- Unwell child
- History of prematurity
- Family history of sudden cardiac death
- History of feeding or respiratory problems
- Social concerns
- Required CPR by medical professional
- Abnormal physical exam

DIFFERENTIAL DIAGNOSES

- Airway obstruction
- Laryngospasm
- Gastroesophageal reflux (GER)
- Apnea (eg. central, apnea of prematurity)
- Congenital heart disease
- Arrhythmia
- Hypoglycemia
- Infection (eg. sepsis, bronchiolitis, pertussis, etc.)
- Seizures
- Inborn errors of metabolism
- Toxic ingestion
- Non-accidental injury

PRESENTATION

- **Event Hx**: duration, color change, change in tone, respiratory effort, relation to feeds, symptoms pre-event, symptoms post-event, resuscitative measures used, sick contacts
- **PMHx**: prematurity, NMS results, development, seizures, reflux
- **FHx**: unexplained deaths, seizures, arrythmias, family member's immunization status
- **SHx**: who lives in the home, who cares for the child, stressors

PHYSICAL EXAM

- **Thorough head to toe physical exam**
- **R/O NAI** – evidence of neglect, bruises to the pinna, neck or abdomen, any injury to the genitalia, fundoscopy, palpation of ribs and long bones

MANAGEMENT OF LOW RISK BRUE

SHOULD	SHOULD NOT	MAY	NEED NOT
Educate caregivers Shared decision-making with parents Resources for caregiver including CPR training	Lab work, lumbar puncture, cultures, CXR, EEG, ECHO, GER studies Home cardiorespiratory monitoring Acid suppression or AED	12-lead ECG Pertussis testing Brief monitoring with continuous pulse oximetry and serial observations	Viral respiratory panel Urinalysis, glucose, bicarb, lactate Neuroimaging Admit to hospital solely for cardiorespiratory monitoring

If there is an explanation or risk factors for a high-risk event, then **investigate accordingly!**

August 2022

Dr. Katharine V. Jensen (Pediatrics Resident, University of Alberta) and Dr. Karen Forbes (Associate Professor of Pediatrics, University of Alberta) for www.pedscases.com

CEREBRAL PALSY

DEFINITION

- Group of non-progressive neurodevelopmental conditions
- Characterized by impairments in motor function that limit activity
- Caused by a disturbance to developing fetal or infant brain

CLASSIFICATION

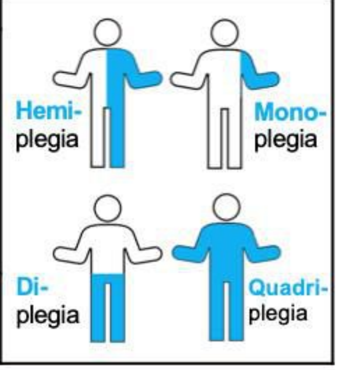
Gross Motor Function Classification System (GFMCS) Levels:

I. Walk without limitations (least functional impact)
II. Walk with limitations
III. Walk using handheld mobility device
IV. Self-mobility with limitations
V. Manual wheelchair (most functional impact)

Motor Types:

spastic, dyskinetic, ataxic, mixed

Affected Limb(s):



DIAGNOSIS

HISTORY

- Inquire about risk factors
- Motor development and milestones
- Comorbidities: epilepsy, learning difficulty, sensory impairments, hip subluxation, scoliosis

PHYSICAL EXAM

- Hypertonia or hypotonia
- Hyperreflexia
- Persistent primitive reflexes: moro, asymmetric tonic neck
- Spasticity

INVESTIGATIONS

1. Neuroimaging (MRI) for correlation with clinical exam
2. If normal MRI, consider metabolic screen and/or genetic testing

CLINICAL RED FLAGS

- Hand preference <12 months
- Stiffness in legs <12 months
- Inability to sit by 9 months
- Makings fists continuously after 4 months
- Other motor or posture delays

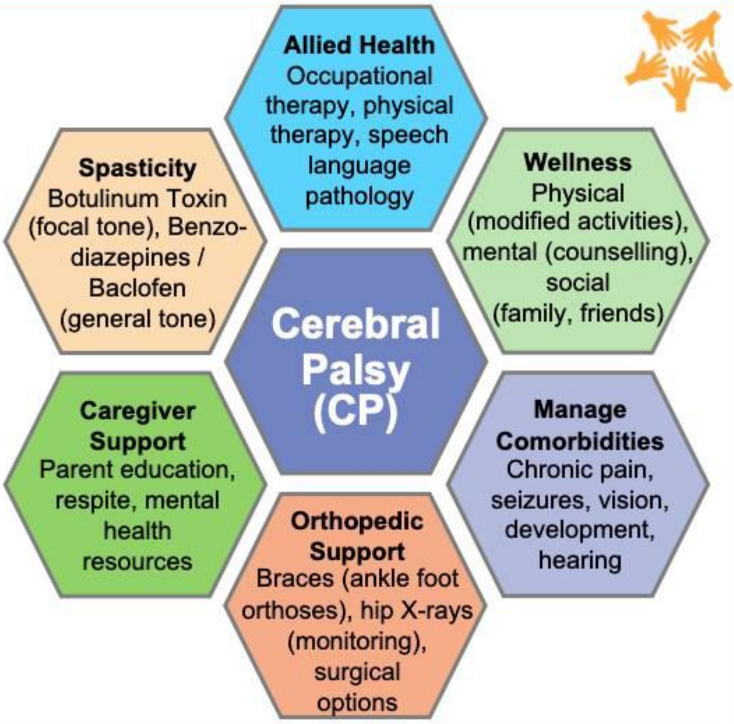
ETIOLOGY

- Genetic
- Birth asphyxia (eg. hypoxic ischemic encephalopathy) (≤10%)
- White matter injury
- Intraventricular hemorrhage
- Perinatal stroke
- Infection (TORCH, meningitis)

SIGNIFICANT RISK FACTORS

- Premature birth
- Low birthweight

SUPPORTIVE MANAGEMENT



Cerebral Palsy (CP)

Allied Health
Occupational therapy, physical therapy, speech language pathology

Spasticity
Botulinum Toxin (focal tone), Benzodiazepines / Baclofen (general tone)

Wellness
Physical (modified activities), mental (counselling), social (family, friends)

Caregiver Support
Parent education, respite, mental health resources

Manage Comorbidities
Chronic pain, seizures, vision, development, hearing

Orthopedic Support
Braces (ankle foot orthoses), hip X-rays (monitoring), surgical options

Published March 2021
Asha Hollis (Medical Student, University of Calgary) and Dr. Scott McLeod (Developmental Pediatrician, Alberta Children's Hospital) for www.pedscases.com

CEREBRAL PALSY

DEFINITION

- Group of non-progressive neurodevelopmental conditions
- Characterized by impairments in motor function that limit activity
- Caused by a disturbance to developing fetal or infant brain

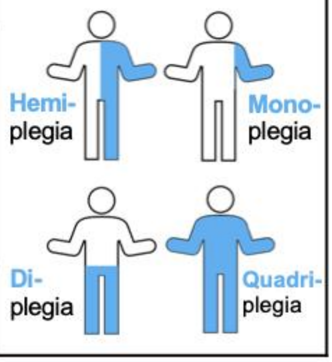
CLASSIFICATION

Gross Motor Function Classification System (GFMCS) Levels:

I. Walk without limitations (least functional impact)
II. Walk with limitations
III. Walk using handheld mobility device
IV. Self-mobility with limitations
V. Manual wheelchair (most functional impact)

Motor Types:
spastic, dyskinetic, ataxic, mixed

Affected Limb(s):



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Published March 2021
Asha Hollis (Medical Student, University of Calgary) and Dr. Scott McLeod (Developmental Pediatrician, Alberta Children's Hospital) for www.pedscases.com

CERVICAL LYMPHADENOPATHY

HISTORY

- **HPI** (lymph node): onset, duration, size, pain, erythema, progression, rate of change, laterality
- **Exposures**: travel, animals/insects, food (unpasteurized dairy, undercooked meat), sick contacts
- **Immunizations** (eg. MMR)
- **IV drug use** and **STIs**

Associated symptoms:

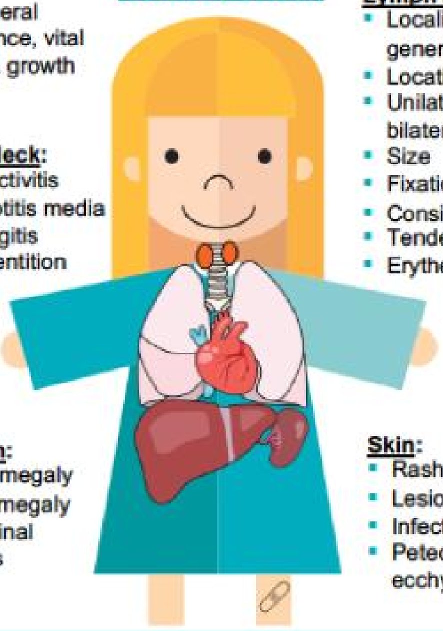
- **Infectious**: pharyngitis, conjunctivitis, rhinorrhea, cough, otalgia, rash, headache, fatigue, myalgia, fevers, chills
- **Autoimmune/inflammatory or rheumatologic**: fatigue, joint pain & swelling, rash, oral ulcers, prolonged or periodic fever, features of Kawasaki disease
- **Malignancy**: weight loss, night sweats, fatigue, pallor, bruising, fever, limp, bone pain

RED FLAGS

Rule out **malignancy** and **chronic infections**.

- Infectious exposures
- Constitutional symptoms
- Generalized adenopathy without evidence of a viral infection
- Progressive painless node enlargement
- Supraclavicular node
- Enlarged node that does not regress/resolve in 4-6 weeks
- Signs of pancytopenia: pallor, bruising, bleeding
- Airway obstruction

PHYSICAL EXAM



General appearance, vital signs, & growth

Head & Neck:

- Conjunctivitis
- Acute otitis media
- Pharyngitis
- Poor dentition

Abdomen:

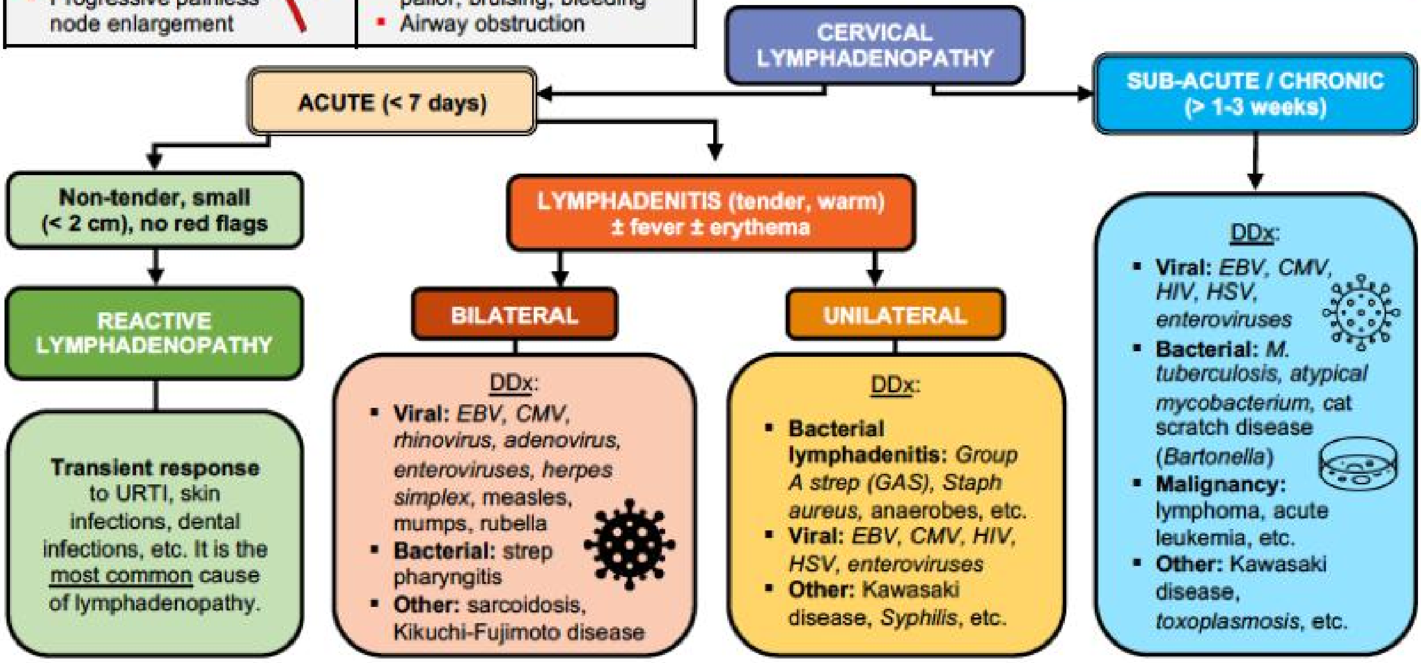
- Hepatomegaly
- Splenomegaly
- Abdominal masses

Lymph Node(s):

- Localized vs. generalized
- Location
- Unilateral vs. bilateral
- Size
- Fixation
- Consistency
- Tenderness
- Erythema

Skin:

- Rashes
- Lesions
- Infection
- Petechiae, ecchymoses



CERVICAL LYMPHADENOPATHY

ACUTE (< 7 days)

Non-tender, small (< 2 cm), no red flags

REACTIVE LYMPHADENOPATHY

Transient response to URTI, skin infections, dental infections, etc. It is the most common cause of lymphadenopathy.

LYMPHADENITIS (tender, warm) ± fever ± erythema

BILATERAL

DDx:

- **Viral:** *EBV, CMV, rhinovirus, adenovirus, enteroviruses, herpes simplex*, measles, mumps, rubella
- **Bacterial:** strep pharyngitis
- **Other:** sarcoidosis, Kikuchi-Fujimoto disease

UNILATERAL

DDx:

- **Bacterial lymphadenitis:** *Group A strep (GAS), Staph aureus*, anaerobes, etc.
- **Viral:** *EBV, CMV, HIV, HSV, enteroviruses*
- **Other:** Kawasaki disease, *Syphilis*, etc.

SUB-ACUTE / CHRONIC (> 1-3 weeks)

DDx:

- **Viral:** *EBV, CMV, HIV, HSV, enteroviruses*
- **Bacterial:** *M. tuberculosis, atypical mycobacterium*, cat scratch disease (*Bartonella*)
- **Malignancy:** lymphoma, acute leukemia, etc.
- **Other:** Kawasaki disease, *toxoplasmosis*, etc.

INVESTIGATIONS

Not all children require investigations.

- **Basics**: CBC & differential, CRP
- **Infectious**: throat culture, serology as indicated (*EBV, CMV, Bartonella*, etc.), TB skin test, blood culture if child is toxic
- **Imaging**: U/S, chest x-ray (for mediastinal mass)
- **Malignancy**: peripheral blood smear, alkaline phosphatase, tumour lysis markers (LDH, urate, PO_4, Ca, K), biopsy

MANAGEMENT

Guided by diagnosis *(not an inclusive list)*

- **Reactive**: observe, re-evaluate in 2-4 weeks
- **Bacterial lymphadenitis**: antibiotics with *Staph aureus* and *Strep pyogenes* coverage
- **Viral**: supportive care
- **Malignancy**: oncology consult
- **Inflammatory**: rheumatology consult, IVIG for Kawasaki disease

Published January 2021
Katharine V. Jensen, Alexander Perry (Medical Students 2021, University of Alberta), and Dr. Hayley Turnbull (Pediatrician & Clinical Lecturer of Pediatrics, University of Alberta) for www.pedscases.com

CHILDHOOD & ADOLESCENT OBESITY

Childhood and adolescent obesity can be defined as a BMI ≥ 95th percentile for age/sex.

Complications of childhood obesity include insulin resistance, type 2 diabetes, non-alcoholic fatty liver disease, obstructive sleep apnea, hypertension, dyslipidemia, slipped capital femoral epiphysis and poor self esteem.

PRESENTATION

HISTORY

Increased weight:
- Weight increasing across ≥ 2 percentile lines on the WHO growth chart between interval visits

Complications of obesity:
- Fatigue/sleep disturbance, joint pain, anxiety/depression, RUQ pain

Reduced linear growth velocity < 5cm/year is abnormal and should raise concern for genetic or endocrine causes of obesity

PHYSICAL EXAM

Growth parameters
- Weight, height on WHO growth charts at every visit
- Plot BMI at least once yearly for children ≥ 2 years
- Waist circumference

Other
- Blood pressure, resting pulse
- Thyroid exam
- Dysmorphic features
- Skin: acanthosis nigricans, striae, hirsutism, acne
- Assess for hypogonadism

DIFFERENTIAL DIAGNOSIS

- ☐ Chronic increase in calorie intake relative to energy expenditure
- ☐ Endocrine disorders: hypothyroidism, Cushing disease, growth hormone deficiency
- ☐ Genetic syndromes associated with obesity: Prader-Willi, Turner, Fragile X, Bardet-Biedel, leptin/leptin receptor deficiency, POMC and MC4R mutations

INVESTIGATIONS

- HbA1c, fasting insulin, LFTs, fasting lipid panel
- AM cortisol, TSH, GH if height velocity slowed
- ECG, echo, sleep study, genetic testing as indicated

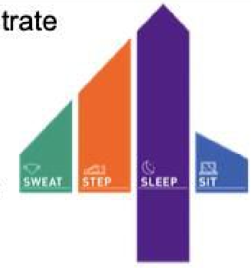
Diagnosis ≥ 2 years

Classification	BMI percentile
Normal	5 ≤ BMI < 85
Overweight	85 ≤ BMI < 95
Obese	≥ 95
Severely obese	BMI ≥ 120% of 95th percentile

Various classification systems exist and definitions vary by age

MANAGEMENT

- **Educate** and **empower parents** using motivational interviewing techniques to demonstrate how the **whole family may achieve a healthier lifestyle.**
- For children ages 5-17, follow the **Canadian 24 hour movement guidelines**. Write a physical activity prescription and encourage more time outside!
- **Reduce sedentary behaviour and screen time** to <2 hours daily for children over 4 years and to <1 hour for children 2-4 years. Screen time for children under 2 years is discouraged.
- Encourage a healthy diet, with reference to the **Canada food guide**. Emphasize the avoidance of sugar-sweetened beverages and casual snacking. Food should not be used as a bribe or reward.
- Referral to a specialized centre for pediatric weight management where the family may access allied health professionals in addition to medical care.
- Manage complications of obesity as indicated.



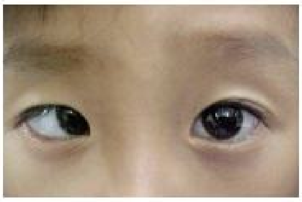
August, 2022
Mikayla Gray (Medical Student, University of Alberta) and Dr. Harvey-Blankenship (Pediatrician, University of Alberta) for www.pedscases.com

CHILDHOOD STRABISMUS

Up to 5% of all children may have some type or degree of strabismus. Strabismus is defined as an abnormal alignment of the eyes. If onset is in childhood, strabismus is often comitant - the misalignment or deviation of the eyes is the same in all gaze positions. Because untreated strabismus is a cause of amblyopia* and irreversible blindness, suspected strabismus should be promptly referred to ophthalmology.

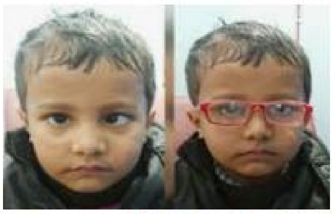
COMMON CHILDHOOD STRABISMUS TYPES AND PRESENTATIONS

1. Congenital esotropia: The constant inward turning of one or both of the eyes. This misalignment of the eyes is often identified prior to 6mo of age.



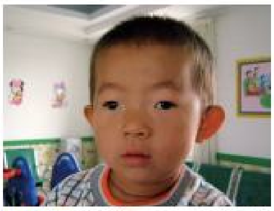
Esotropia © Community Eye Health, CC BY-NC 2.0

2. Accommodative esotropia: The inward turning of one or both of the eyes when trying to focus on close objects. Onset is typically between 6mo-7yrs of age and starts as an intermittent strabismus that becomes constant with time.



Accommodative Esotropia © IAPB/VISION, CC BY-NC-SA 2.0

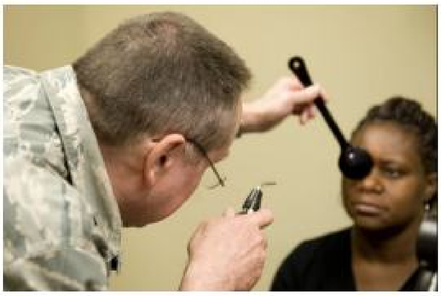
3. Congenital exotropia: The constant outward turning of one or both of the eyes. Typical presentation is before 6mo of age and is associated with craniofacial malformations and neurologic abnormalities.



Exotropia © Community Eye Health, CC BY-NC 2.0

PHYSICAL EXAM: TESTS THAT HIGHLIGHT STRABISMUS

- Ophthalmoscopy and Red Reflex (the misaligned eye may have a brighter and lighter red reflex)
- Corneal Light Reflex (light may not be centered on the pupil of the deviated eye)
- Cover Test (if the uncovered eye moves to pick up fixation, this suggests a strabismus)
- Extraocular eye movement testing (misalignment of the eye may be observed in all gaze positions)



Cover Test © 176th Wing, Alaska Air National Guard, CC BY 2.0

***Amblyopia** is a decrease in visual acuity due to a disruption in how the brain and eyes communicate. Strabismus is the most common cause of amblyopia.This disruption can cause a progressive decrease in visual acuity with varying degrees of correctability.

NEXT STEPS

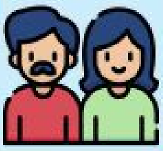
If strabismus is suspected, ophthalmology needs to be consulted.

November 2023

Megan Pohl (Medical Student, University of Alberta), Christopher Rudnisky (MD MPH FRCSC, Department of Ophthalmology & Visual Sciences, University of Alberta)

CIRCUMCISION

INFORM



"Should we circumcise our baby boy?"

Circumcision: permanent surgical removal of the prepuce (foreskin), a fold of tissue that covers the glans (head) of the penis.

Elective circumcisions are NOT recommended as routine neonatal intervention in Canada

Canadian Pediatric Society (CPS): due to **close balance of medical risk-to-benefit ratio**, the informed decision of circumcision should be **made by parents after weighing individual risks and benefits** to their son in context of their familial, religious, and cultural beliefs.

Phimosis (non-retractile foreskin) = inability of the prepuce to retract back over the glans

Physiologic phimosis: foreskin present at birth is **normally non-retractile!** Congenital adhesions between glans and prepuce will **loosen by puberty** in majority of cases, allowing for eventual retraction.

Pathologic phimosis: **problematic** scarring of prepuce that develops later in life due to infection and inflammation.

- **First line** treatment: **topical steroid cream** (0.05 – 0.1% betamethasone) BID with gentle traction. Successful in 80%of cases
- If unsuccessful, should consider **medically necessary** circumcision.

History and Physical Exam

- Impaired urinary stream? (weak, dribbling, spraying)
- Recurrent UTIs?
- Unable to visualize urethral meatus?
- Blanched patches? (Indicative of lichen sclerosis)

Contraindications:

Congenital penile **abnormalities** such as

- Hypospadias
- Dorsal hood deformities
- Chordee
- Micropenis
- Webbed penis
- Buried penis
- Large hydrocele or hernia

Refer to pediatric urology

Delay in unstable or premature infants

Relative contraindications: bleeding disorders

DISCUSS

Benefits:

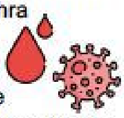
- ✓ Prevents **paraphimosis** (painful retraction of the foreskin that cannot be returned to normal position)
- ✓ Reduced risk of **balanitis** (inflammation of the glans)
- ✓ Lower rate of **pathologic phimosis** (NNT* = 67)
- ✓ Reduced rate of **UTIs** in 1st year of life (NNT = 125 in normal infant boys; NNT = 5 in high risk groups such as recurrent UTI or underlying urinary tract anomaly)
- ✓ Reduced rate of **HSV** infection (NNT = 16)
- ✓ Reduced rate of **HPV** infection (NNT = 5) and reduced risk of cervical cancer in female partners (NNT = 120)
- ✓ **No** negative impact on sexual function or satisfaction in males or their partners.

***NNT** = number needed to treat

Risks:

Surgical Risks:

- X Minor: local infection, minor bleeding
- X Severe: sepsis, major hemorrhage, permanent anatomical damage to glans or urethra
- X Late: meatal stenosis, penile adhesions, buried penis
- X Unsatisfactory cosmetic appearance
- X Anesthetic risks if requiring general anesthesia



Complication rates:

- X 1.5% in neonates and infants
- X 6% in childhood, adolescence, and adulthood

Ethical concerns of child being unable to consent

DECIDE

- ☐ Guide parents in **informed decision making** by educating about all known risks and benefits
- ☐ If unsure, offer online resources and schedule for follow up
- ☐ **Be mindful** that this procedure has important religious, cultural, and ethnic significance, and acknowledge these values in the decision making process

caringforkids.cps.ca

"circumcision"

"Okay, let's go ahead"

- **Refer** parents to a medical professional (pediatrician, pediatric urologist, family doctor) who is **trained and well practiced** in the procedure to **significantly decrease complication rates.**
- Various procedure methods such as freehand technique or device-assisted have similar outcomes.
- Generally recommended to undergo procedure during infancy (< 2 weeks of age) using only local anesthesia. If done at older age the procedure is associated with higher complication rates and requires general anesthesia.

"No, not for us"

- **No special care needed** for uncircumcised penis. Routine bathing and hygiene practices are sufficient.
- **Do not forcibly retract the foreskin** as this may cause scarring, leading to adhesions.
- Only 4% of newborn males will have a completely retractable foreskin at birth; **50%** by **6** years old; and **95%** by **17** years old.
- As the male ages, growth of the penis and physiologic erections will loosen adhesions, allowing for retraction.
- If the foreskin has been retracted, **always replace back** over the glans to prevent paraphimosis.

Published December 2021

Sarah Park (Medical Student C2022, University of Alberta), Dr. Peter Metcalfe (Pediatric Urologist, University of Alberta) for www.pedscases.com

CLEFT LIP AND PALATE

BACKGROUND

- **Cleft lip and palate (CL/P)** is a subcategory of orofacial clefts (OFC)
- OFCs also include: isolated cleft palate and isolated cleft lip
- Oral facial clefts occur in 1:750 live births
- Cleft lip and palate is more frequent in males (2:1) and the most common configuration is a left unilateral CL/P

EMBRYOLOGY

- Failure of the medial nasal process to contact the maxillary process results in cleft lip [weeks 4-7 of gestation]
- Failure of fusion of the palatine shelves with the primary palate and/or failure of fusion of palatine shelves together with the nasal septum results in cleft palate [weeks 4- 12 of gestation]

ETIOLOGY

Environmental factors associated with an increased risk:

- Maternal smoking
- Maternal corticosteroid use
- Teratogens

Genetic factors:

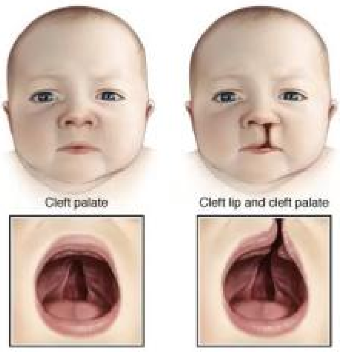
- **Non-syndromic:** Combination of multiple interacting genes and multifactorial inheritance pattern
- **Syndromic:** Cleft lip and/or palate is associated with ~400 genetic syndromes

DIAGNOSIS

- Clefts of the lip and palate are typically recognizable upon newborn exam
- Clefts of the submucous cleft palate may not be identified on newborn examination and may present later in a child having difficulty with feeding, swallowing, chronic ear infections, and/or hyper nasal voice

INVESTIGATIONS

- Second-trimester prenatal ultrasound has an 88% sensitivity for detection of CL±CP
- Prenatal genetic screening
- Newborn physical examination of face, mouth, nose, and palate



Associated Features	PATHOPHYSIOLOGY	MANAGEMENT
Feeding	Clefts of the palate make sucking difficult as they are not able to generate suction and have oronasal communication	• Specialist feeding assessment • Monitoring of weight gain/ growth • Swallowing assessments • Special CL/P bottle/nipple, nasogastric tube placement (rare)
Ear Disorders	Infants with cleft palate are at an increased risk of developing conductive hearing loss, middle ear disease and eustachian tube dysfunction	• Otolaryngologist and audiologist referral • Hearing screen and hearing assessment
Speech	Due to the lack of normal function of the palate, phonetic errors may arise and oronasal communication may lead to hyper nasal speech	• Speech-language pathologist referral • Regular assessment of speech
Oral Health	Children with a CL/P often have missing, extra and/or malformed teeth	• Dentist and orthodontist referral • Regular dental assessment

MANAGEMENT

Surgery is the primary treatment for CL/P

- **Timing: Cleft palate repair:** 9 – 12 months (<12 mo. important to facilitate normal speech development)
- **Cleft lip repair:** 3- 6 months

Goals: Return of normal function and form of lip, nose, and palate

Specialist: Craniofacial surgeon

August 2023
Emma Yanko (Medical Student, University of Saskatchewan), Dr. Craig Gendron (Pediatric Plastic Surgeon, University of Saskatchewan) for www.pedscases.com

PAIN & DISTRESS MANAGEMENT FOR BRIEF MEDICAL PROCEDURES

COMMON PAINFUL BRIEF MEDICAL PROCEDURES

- Intravenous cannulation
- Blood draws
- Heel lances
- Lumbar puncture
- Intramuscular injections
- Urine collection in infants
- Abscess drainage
- Dressings for burns
- Nasogastric tube insertion
- Laceration repair
- X-ray for fractures/dislocations

Choose the most **minimally invasive** approach possible. Always try to **combine preventative, physical, psychological,** and **pharmacological approaches** to pain and distress management.

PREVENTION

Whenever possible:

- Choose venipuncture over heel lances or IM injections.
- Couple IV insertion together with blood tests.
- Daily bloodwork should not be prescribed in an automatic, recurring fashion.
- Group blood tests together.

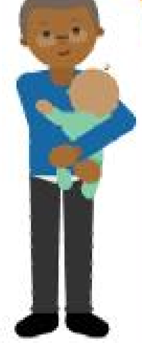
PSYCHOLOGICAL

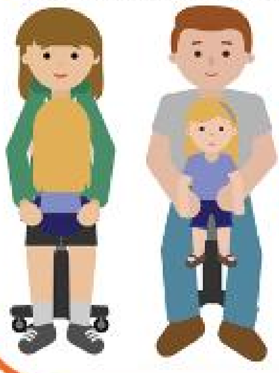
- **Caregiver** presence and involvement
- Use **calm language**
- **Avoid false or premature reassurance**. Don't say things like *"this won't hurt", "it's almost over"*.
- **Age-appropriate preparation**: can show younger children medical supplies to familiarize them with the equipment; for children ≥ 4 years, explain the procedure in a developmentally-appropriate way. Offer them realistic choices.
- **Child-friendly environment**: toys, wall decor, pictures
- **Distraction**:
  - Involve child life therapist, if available
  - Engage the child in non-procedural conversation
  - Bubbles, reading, videos, games, music
  - Deep breathing

PHYSICAL

Infants:

- Pacifier
- Swaddling
- Tucking
- Rocking
- Breastfeeding or sucrose
- Skin-to-skin (kangaroo) care





Children:

- Sitting on caregiver's lap
- Comfort holds
- Sitting upright, rather than lying down, enhances the child's sense of control

PHARMACOLOGICAL

Topical anesthetics:

- Liposomal lidocaine (Maxilene)
- Amethocaine (Ametop)
- Lidocaine-prilocaine (EMLA)
- Vapo-coolant spray (Pain Ease)
- LET gel (lidocaine 4%/ epinephrine 0.1%/ tetracaine 0.5%)

Local anesthetics:

- Lidocaine 1%
- Nerve block

Systemic:

- **Ibuprofen** is superior to acetaminophen for acute pain.
- Ibuprofen can be used for mild to moderate pain or as co-analgesia for moderate to severe pain.
- **Nitrous oxide** – mild sedation & analgesia offered for older children, for some procedures.
- **Intranasal fentanyl** for moderate to severe pain.

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Katharine V. Jensen (Medical Student 2021, University of Alberta), Dr. Evelyne Trottier (Université de Montréal), Dr. Samina Ali (University of Alberta), and Dr. Hayley Turnbull (University of Alberta) for www.pedscases.com. Images provided by ECHO Research, University of Alberta (Dr. Shannon Scott, PI).

COMMUNITY-ACQUIRED PNEUMONIA (CAP)

Key Points

- Non-specific presentation, but common clinical features include:

Acute fever | Cough | Short of breath | Vomiting

Physical exam: tachypnea | lung crackles | percussion dullness | ↑ tactile fremitus | ↓ vesicular & ↑ bronchial breath sounds

- Confirm diagnosis with **chest X-ray (CXR)**

Pathogens

- **Typical** bacterial → ***Streptococcus pneumoniae*** **(most common)**
  - **Consolidation** on CXR supports diagnosis
- **Atypical** bacterial (*Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*)
  - Bilateral **focal** or **interstitial infiltrates** on CXR supports diagnosis
- **Viral** (eg. RSV, influenza, hMPV) → most common etiology <5 years of age
  - Patchy **infiltrates** or **atelectasis** on CXR supports diagnosis

Management

Empiric **amoxicillin** 40-90 mg/kg/d PO (up to 4 g/day) in 3 divided doses × 7-10 days

if suspect/detect…

Atypical bacterial CAP

Azithromycin (up to 500 mg/day)
- ✓ Day 1: 10 mg/kg/d IV/PO
- ✓ Days 2-5: 5 mg/kg/d IV/PO

Viral CAP (influenza season)

If symptoms <48 hrs or moderately/severely ill, consider: neuraminidase inhibitors (**oseltamivir, zanamivir**)

Consider hospitalization & IV antibiotics if:

hypoxic | inadequate oral intake | intolerant of oral therapy | severe illness | or complex CAP

March 2020
Richard He (MD Student 2022, University of Alberta) for www.pedscases.com

COMPLETE TRANSPOSITION OF THE GREAT ARTERIES (D-TGA)

Transposition of the great arteries (TGA) is a congenital heart defect where the aorta and pulmonary artery are switched in position. This results in **parallel circulations**, where oxygenated blood circulates to lungs, and deoxygenated blood circulates to the body.

PRESENTATION

- **Epidemiology**
  - ~ 20% of all cyanotic CHD
- **Associated cardiac defects**
  - VSD, LVOTO, abnormal valves and/or coronary arteries
- **Etiology**
  - Multifactorial/unknown
  - Infants born to mothers with diabetes
  - DiGeorge Syndrome (22q11 deletion)

PHYSICAL EXAM

General Exam

- Persistent cyanosis from birth
- Tachypnea
- Decreased SpO2

Cardiac Exam

- Single, loud S2
- Usually no murmur
- Diminished femoral pulses if associated with coarctation or heart failure

PATHOPHYSIOLOGY

- Right ventricle connected to aorta, and left ventricle connected to pulmonary artery □ complete isolation of pulmonary and systemic circuits
- Two main forms of TGA
  - D-TGA (most common) – ventricles in normal positions, aorta and pulmonary artery reversed
  - L-TGA – ventricular positions reversed (ventricular inversion), as well as aorta and pulmonary artery being reversed (systemic venous drainage directed to pulmonary artery, and pulmonary venous drainage directed to the aorta as normal)

Patent ductus arteriosus (PDA) (or large ASD or VSD) critical to prevent severe cyanosis and decompensation

DIAGNOSIS/INVESTIGATIONS

Prenatal Diagnosis

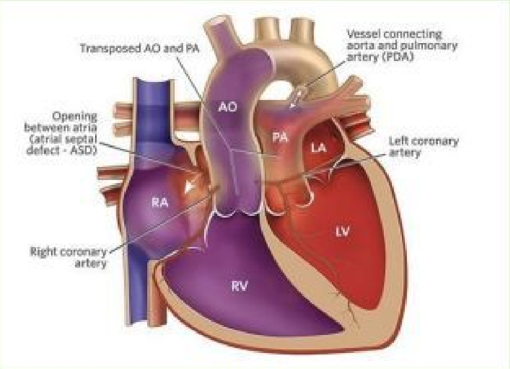
- ❑ TGA can be difficult to diagnose with fetal ultrasound

Imaging

- ❑ Echocardiography (confirmatory test)
  - Pulmonary artery arising from left ventricle, and aorta arising anteriorly from right ventricle
  - Assess for associated ASD, VSD, or PDA
- ❑ Chest X-Ray
  - **"Egg on a string"** appearance of heart

Other

- ❑ ECG – often normal, may have RVH or RAD
- ❑ Hyperoxia test – **positive** – decreased SpO2, unresponsive to supplemental oxygen


Transposed AO and PA
Vessel connecting aorta and pulmonary artery (PDA)
Opening between atria (atrial septal defect - ASD)
AO
PA
LA
Left coronary artery
RA
Right coronary artery
LV
RV

D-TGA

MANAGEMENT

Initial postnatal management = ensure mixing between systemic and pulmonary circulations

- □ Infusion of prostaglandins (PGE1) – prevents closure of PDA
- □ Balloon Atrial Septostomy – right heart catheterization to create ASD or enlarge existing ASD

Surgical repair – recommended within first 10 days of life

- □ Arterial Switch Procedure – transecting and connecting aorta to the left ventricle and pulmonary artery to the right ventricle with coronary artery transfer to the neo-aorta being the previous pulmonary artery

September 2024

Lize-Mari Du Toit (Medical Student, University of Alberta), Michal Kantoch (Pediatric Cardiologist, University of Alberta) for www.pedscases.com

COMPLICATIONS OF PRETERM BIRTH (PTB)

Definition of PTB: < 37^{+0} wks
Late preterm: 34^{+0} – 36^{+6} wks
Extremely preterm: < 28^{+0} wks

INITIAL ASSESSMENT

Does the baby…

1. appear full term? (Ballard Scale)
2. have good muscle tone?
3. appear to be breathing or crying?

If **no** to any □ stimulate, position airway, suction, and provide ventilatory support as needed.

ETIOLOGY

Maternal factors	Placental/Uterine factors	Fetal factors
▪ History of prior preterm delivery ▪ Extremes of age (< 17 y.o., >35 y.o.) ▪ Medical conditions (hypertension, diabetes) ▪ Substance use/smoking	▪ Intrauterine stretch (i.e multiple gestation) ▪ Infection (i.e. chorioamnionitis, bacterial vaginosis) ▪ Placental insufficiency ▪ Uterine anomalies	▪ Congenital anomalies (i.e. CNS defects) ▪ Intrauterine growth restriction ▪ Fetal distress

SYSTEM	PRESENTATION	MANAGEMENT
Respiratory	**Respiratory Distress Syndrome:** lack of surfactant □ atelectasis	Antenatal corticosteroids as prophylaxis. Surfactant and respiratory support.
	Apnea of Prematurity: episodes of apnea > 20s or shorter if associated with change in vitals due to immature respiratory control	Caffeine maintenance. Stimulation/Respiratory support during episodes. Self-resolving by 37 weeks.
Cardiac	**Patent Ductus Arteriosus:** Delayed closure of fetal shunt, leads to systemic hypoperfusion	Ibuprofen/acetaminophen/indomethacin to induce closure. Fluid restriction. Surgical ligation if needed.
GI	**Necrotizing Enterocolitis:** Necrosis of intestinal mucosa due to infection. Presents with feeding intolerance, bloody stool, obstruction and/or perforation	NPO, Gastric decompression, IV Fluids, IV antibiotics, surgery if evidence of perforation.
Brain	**Intraventricular Hemorrhage:** Intracranial bleeding, multifactorial causes	U/S screening if <32 wks, serial monitoring. Neurosurgical consult if abnormal.
Eye	**Retinopathy of Prematurity:** proliferation of retinal blood vessels. If untreated, can cause blindness	Screening if ≤ 30 wks GA. Laser photocoagulation and anti-VEGF (vascular endothelial growth factor).
Immune	**Sepsis:** Most commonly due to Group B Strep and E. Coli	IV antibiotics; supportive care
Blood Tests	**Hypoglycemia** **Anemia** **Hyperbilirubinemia**	**Hypoglycemia:** Increase feeds, IVF, TPN **Anemia:** Iron supplements, transfusions **Hyperbilirubinemia:** Phototherapy

July 2024
Ola Elsharif (Medical Student) and Dr. Meenatchi Ranganathan (General Pediatrician) McMaster University
for www.pedscases.com

CONGENITAL STORCH INFECTIONS

Congenital STORCH infections = infection in the **fetal period** by one of a group of pathogens that cause a **similar constellation of clinical features** after intrauterine transmission.

Symptomatology depends on **timing of transmission** during pregnancy.

INITIAL EVALUATION

- ☐ History and physical examination
- ☐ Labs: CBC, LFTs
- ☐ Imaging: head and abdomen
- ☐ Referrals: audiology, ophthalmology
- ☐ +/- Lumbar puncture

PRESENTATION

Confirmed intrapartum maternal STORCH infection

or

Fetal clinical presentation

- Microcephaly, intracranial calcifications, hydrocephalus, seizures, lethargy, fever
- Chorioretinitis, cataracts, microphthalmia
- Sensorineural hearing loss
- Feeding difficulties, hepatosplenomegaly, elevated transaminases
- Congenital heart disease, myocarditis
- Thrombocytopenia, anemia
- Purpuric rash, erythematous rash, jaundice
- IUGR, hydrops fetalis, preterm labor, intrauterine demise

	S	T	O (Other)		R	C	H
Pathogen	**Syphilis**	**Toxo–plasmosis**	**Varicella Zoster Virus (VZV)**	**Parvovirus B19**	**Rubella**	**Cytomega-lovirus (CMV)**	**Herpes Simplex Virus (HSV)**
Prenatal screening	Maternal syphilis EIA and RPR	None	Maternal VZV IgG	None	Maternal rubella IgG	None	Maternal skin lesion swab PCR
Initial diagnostic tests	Infant syphilis serology & RPR	Infant toxoplasma IgG, IgM, IgA	Infant VZV IgG, IgM & lesion PCR	Infant parvovirus IgM, IgG & serum PCR	Infant rubella IgM, IgG & urine / throat PCR	Infant urine / saliva PCR	Infant serum / CSF/ skin PCR

MANAGEMENT

- Early family counselling on possible long-term sequelae.
- Early involvement of Peds ID.

In utero

Involve **maternal-fetal specialists** to follow baby's development.

Birth

Skilled **NICU ready** for transfer. Care team at delivery.

Infant

Supportive treatment. Specific treatment once pathogen known.

Published January 2022
Terra Morel (Medical Student, University of Alberta) and Dr. Sarah Forgie (Professor, Pediatric Infectious Disease, University of Alberta) for www.pedscases.com

CONJUNCTIVITIS

"Pink eye". Inflammation of the conjunctiva: the transparent tissue covering the outer wall of the eye (sclera) and the inner surface of the eyelids. Dilation of conjunctival blood vessels causes the pink hue.

PRESENTATION

	ALLERGIC	IRRITANT	VIRAL	BACTERIAL
Laterality	**Bilateral**	**Bilateral**	Bilateral	**Unilateral** > bilateral
Discharge	Minimal, ropey	Watery	Minimal, watery	**Purulent**
Sensation	**Itchy**, gritty, blurriness	**Burning**, gritty, blurriness	Irritation, stinging, blurriness	Irritation, stinging, blurriness
Associated with	▪ Puffy "allergic shiners" ▪ **Allergen exposure**	▪ Chlorine, shampoo, smoke, chemicals	▪ Pre-auricular lymphadenopathy ▪ **URTI** symptoms ▪ Sick contacts	▪ Sick contacts ▪ Poor contact lens hygiene

MANAGEMENT

	ALLERGIC	IRRITANT	VIRAL	BACTERIAL
Cool compress, Artificial tears	✓	✓	✓	✓
Antihistamines	✓			
↓ Exposure	✓	✓		
Frequent hand hygiene			✓	✓
No contact lenses until resolved	✓	✓	✓	✓
Topical antibiotics for bacterial *only*	**Non-contact lens users**: Erythromycin or Bacitracin/Polymyxin B ● Covers: *H. influenzae, S. pneumoniae, M. catarrhalis, Staphylococcus spp.* **Contact lens users**: fluoroquinolones (Moxifloxacin or Ciprofloxacin) ● Covers: ***Pseudomonas*** in addition to the above			

Red flags for urgent ophthalmology referrals

HSV infection	**Unilateral vesicular rash** or ++ pain	Empiric acyclovir
Gonococcal	Hyperacute, **hyperpurulent**, severe	Empiric ceftriaxone + azithromycin
Ophthalmia Neonatorum	Conjunctivitis in ≤30 days of life	All the above to cover HSV and CT/NG

October 2023

Kimberly Papp (Medical Student C2024, University of Alberta), Dr. Matthew Benson (Ophthalmologist, University of Alberta) for www.pedscases.com

CONSTIPATION

Constipation: infrequent, difficult, painful, or incomplete evacuation of hard stools

CAUSES OF CONSTIPATION

Organic:
- Hirschsprung disease
- Hypothyroidism
- Cystic fibrosis
- Celiac disease
- Diabetes
- Spinal cord abnormalities
- Anorectal malformation
- Drugs (eg. opioids, antidepressants)
- Toxins (eg. lead)

Functional (90-95%):
- Irritable bowel syndrome
- Withholding behaviour (eg. fighting the urge to go due to fear or embarrassment)
- Poor fluid intake
- Insufficient dietary fiber
- Dietary changes
- Sedentary lifestyle

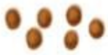
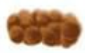
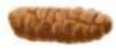
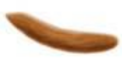
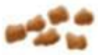
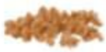
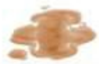
PRESENTATION

- Infrequent stools (<3x/week)
- Hard or painful to pass stool
- Large, difficult to flush stool
- Blood in the stool
- Fecal incontinence (encopresis)
- Overflow diarrhea
- Abdominal pain or cramps
- Difficulty urinating
- Behavioural issues

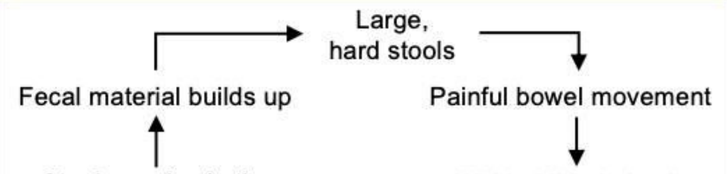
PHYSICAL EXAM

- Abdominal exam: distension, bowel sounds, palpation for masses and tenderness
- Anal fissures
- DRE to check for rectal tone, stool mass, and stool consistency
- Features of spina bifida (sacral dimple, hair tuft)
- Misplaced anal opening

BRISTOL STOOL CHART

TYPE 1		**Severe constipation** – separate hard lumps, difficult to pass
TYPE 2		**Mild constipation** – sausage shaped, but lumpy
TYPE 3		**Normal** – like a sausage, but with cracks on the surface
TYPE 4		**Normal** – like a sausage or snake, smooth and soft
TYPE 5		**Lacking fiber** – soft blobs with clear-cut edges (passes easily)
TYPE 6		**Mild diarrhea** – fluffy pieces with ragged edges, a mushy stool
TYPE 7		**Severe diarrhea** – watery, entirely liquid, no solid pieces

PATHOPHYSIOLOGY OF FUNCTIONAL CONSTIPATION



Large, hard stools

Painful bowel movement

Withholding behavior due to pain or inconvenience

Stool remains in the colon → colonic reabsorption of water

Fecal material builds up

INVESTIGATIONS

No investigations needed if history and physical exam are consistent with functional constipation

Investigations to consider:
- Abdominal x-ray, TSH, celiac testing, HgA1C, CF testing, electrolytes, extended electrolytes, lead level

Red flags: delayed passage of meconium (> 48 hours), young infants, poor feeding and/or growth, recurrent fevers, bilious emesis, bloody stool, developmental delays, hypotonia, poor suck, abnormal neurologic exam

MANAGEMENT

Disimpaction
Bowel clean out with PEG3350 or Pico-salax

Maintenance
PEG3350 for ≥ 3 months to restore normal bowel routine. Titrate to effect.

Regular toileting
Have the child sit on the toilet after each meal.

Healthy diet

Increase fluid intake

Increase physical activity

Arrange **follow-up**

June 2022
Dr. Katharine V. Jensen (Pediatrics Resident, University of Alberta) and Dr. Jason Silverman (Associate Professor, Department of Pediatrics, University of Alberta) for www.pedscases.com

CONTRACEPTION CARE FOR YOUTH

Almost 20,000 young women (< 20 years of age) get pregnant each year and 70% of those pregnancies are unintentional. By assisting youth to access effective contraception and use it consistently, we can help reduce unintentional pregnancies.

HISTORY

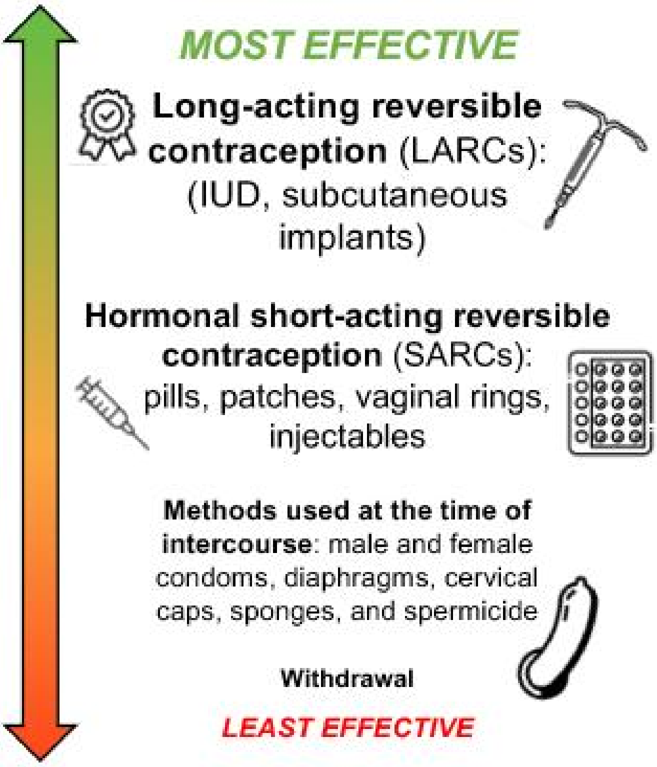
- Discuss **sexual health**, **family planning**, and **contraception** with all youth – even those who are not yet sexually active.
- Screen the **past medical history**.
- Look up **medications** and **medical conditions** in the World Health Organization Medical Eligibility Criteria (WHO-MEC) to determine if there is a contraindication to contraception.
- Contraindications are rare in youth and **most teens can safely start contraception**.

CONTRACEPTION COUNSELLING

- **Recommend contraception in order of effectiveness** as it influences the patient's choice. **LARCs are 1st line!**
- LARCs are **safe in youth**.
- Discuss the **effectiveness, risks,** and **side effects** of each method.
- **Prescribe collaboratively** by involving youth in the decision of which contraceptive is best for them and respect that decision.
- Encourage **adjunctive condom use** to prevent transmission of STIs.
- Inform all youth about **emergency contraception options**.

PHYSICAL EXAM

- **Blood pressure**
- A pelvic exam is **NOT** required for contraception.
- **STI screening is encouraged,** but not required for the initiation of contraception.
- **Screen for STIs** either before or at the time of IUD insertion.



MOST EFFECTIVE

Long-acting reversible contraception (LARCs): (IUD, subcutaneous implants)

Hormonal short-acting reversible contraception (SARCs): pills, patches, vaginal rings, injectables

Methods used at the time of intercourse: male and female condoms, diaphragms, cervical caps, sponges, and spermicide

Withdrawal

LEAST EFFECTIVE

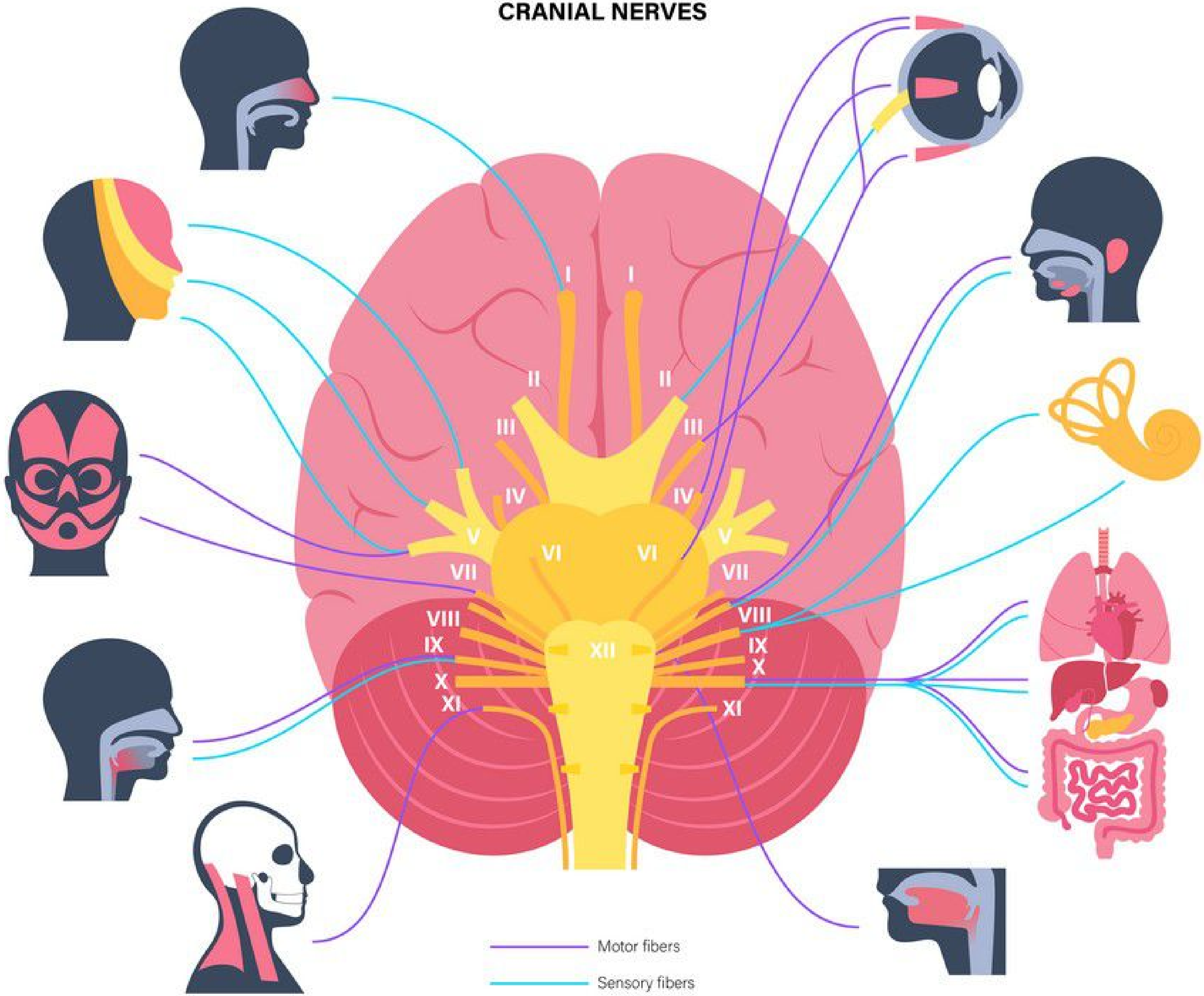
REMOVE BARRIERS TO CONTRACEPTION

- **No need for a separate appointment**, provide a prescription for contraception whenever youth request it.
- **No need for a pelvic exam**, except at the time of IUD insertion.
- **No need for STI testing**, except at the time of IUD insertion.
- **Quick start**
- Provide **long-term 12-month prescriptions**
- Ask about **how they will pay** for the contraceptive and help them figure out where/how to get it, especially if they need it confidentially.

September 2020

Katharine V. Jensen (Medical Student 2021, University of Alberta) & Dr. Giuseppina Di Meglio (Associate Professor of Pediatrics, Montreal Children's Hospital/McGill University Health Center) for www.pedscases.com

CRANIAL NERVES


I
I
II
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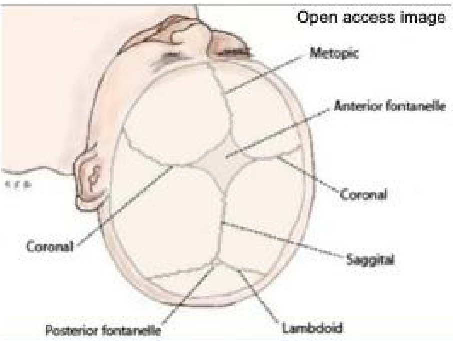
V
V
VI
VI
VII
VII
VIII
VIII
IX
IX
XII
X
X
XI
XI
Motor fibers
Sensory fibers

CRANIOSYNOSTOSIS

Craniosynostosis:
premature fusion of 1+ cranial sutures; this is *always abnormal*
Functional sequelae:
craniofacial deformity, restricted brain growth, elevated ICP, ophthalmic anomaly, Chiari malformation

PATHOPHYSIOLOGY

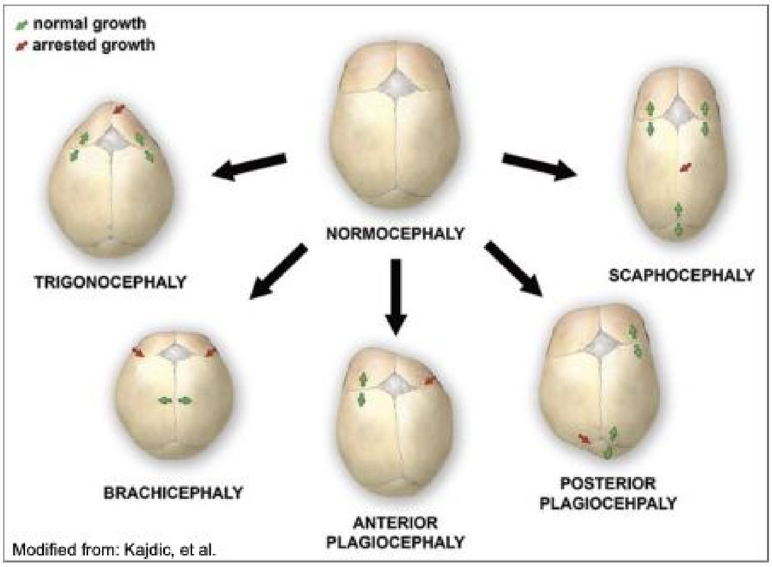
- **Cranium growth:** occurs through *sutural growth* (perpendicular to cranial sutures) and *appositional growth* (resorption of bone from the inner cranial surface & deposition on the outer surface)
- **Virchow's Law:** premature suture fusion results in cranial growth predominantly parallel to fused sutures
- **Causes**: intrauterine compression, syndromic, endocrinopathy, cranial base anomaly, abnormal dural attachment, medications


Open access image

The resulting craniosynostosis is dependent on suture(s) involved

PRESENTATION

HISTORY	PHYSICAL EXAM
Onset & evolution • At birth or early acquired **Positional preference** **Torticollis** **Signs of elevated ICP** • Lethargy, irritability, poor feeding, nausea, vomiting visual changes, altered mentation, discoordination	**Inspection** • Abnormal cranial morphology or facies • Syndromic features **Palpation** • Ridge or lack of movement at suture, bulging fontanelles **Head Circumference**



Modified from: Kajdic, et al.

DIFFERENTIAL DIAGNOSIS
(abnormal head shapes not caused by suture fusion)

Deformational plagiocephaly: parallelogram skull shape with anterior displacement of ipsilateral occiput, ear, forehead
Deformational brachycephaly: shortened AP distance and flattened occiput with normal forehead and orbits
Dolichocephaly: elongated AP distance without biparietal narrowing or frontal/occipital bossing; common in NICU
• *Distinguish by positional preference, movement at suture, lack of palpable sutural ridge, no functional sequelae, imaging*

MANAGEMENT

Consult: to pediatric craniofacial surgeon. If signs of elevated ICP, consult neurosurgery (do not delay!)
Imaging: radiographs of skull (AP, lateral, Towne's projection) and C-spine (for associated abnormalities)
• Discuss with craniofacial surgeon if low dose CT indicated for complex diagnosis and/or surgical planning
Surgery: ~1st year; technique depends on timing & specific features; +/- helmet therapy
Multidisciplinary: neurosurgery, ophthalmology, speech language pathology, occupational therapy
Prognosis: variable based on craniosynostosis +/- associated syndrome

October 2023
Colborne Kemna (Medical Student, University of Alberta), Dr. Alexander Perry (Plastics Resident, University of Alberta) & Dr. Curtis Budden (Assistant Professor of Plastic Surgery, University of Alberta) for www.pedscases.com

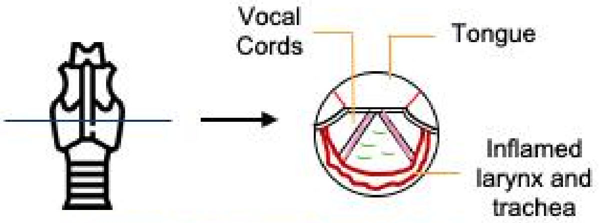
ACUTE MANAGEMENT OF CROUP

Mean age of diagnosis

3 months — **18 months** — 3 years

CAUSATIVE ORGANISMS

Parainfluenza (65%)
Influenza A
Influenza B
Adenovirus
RSV
Metapneumovirus



Laryngotracheobronchitis

Croup is a clinical diagnosis. Radiographs are not routinely recommended. The characteristic "steeple" sign is only present in 50% of cases with croup. The "steeple sign" can also be present in children without croup depending on their phase of respiration at the time of radiograph

TREATMENT AND MANAGEMENT

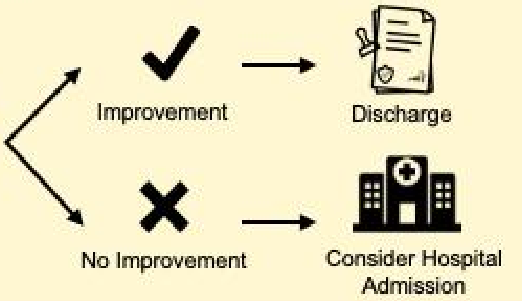
	Mild	Moderate	Severe
Barky "Seal Like" Cough	Occasional	Frequent	Frequent
Stridor	Minimal	At Rest	Prominent
Respiratory Distress	None	Indrawing	Severe Indrawing
Lethargy or Agitation	None	Limited	Substantial

MILD

1 Dose of Oral Dexamethasone **0.6mg/kg** → Discharge

MODERATE

1 Dose of Oral Dexamethasone **0.6mg/kg** → Observe for 4 Hours



✓ Improvement → Discharge

✗ No Improvement → Consider Hospital Admission

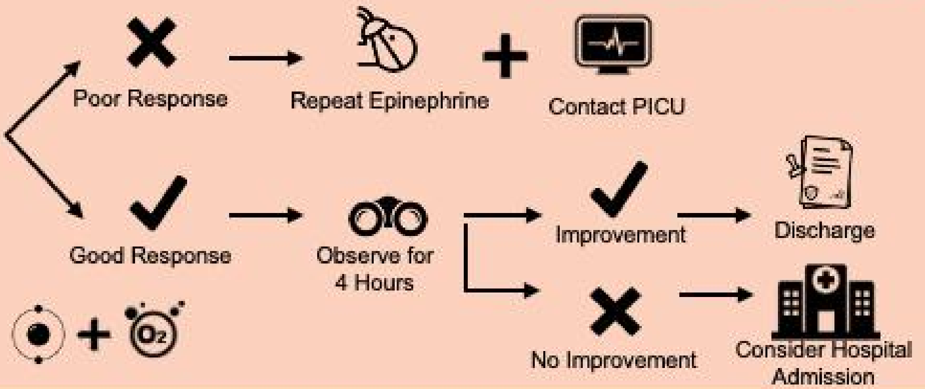
CRITERIA FOR ADMISSION

If after **four hours** post steroids:

1. Moderate Respiratory Distress
2. Stridor at Rest
3. Chest Wall Indrawing

SEVERE

1 Dose of Oral Dexamethasone **0.6mg/kg** + Nebulized Epinephrine **L Epinephrine 1:1000 5mL**



✗ Poor Response → Repeat Epinephrine + Contact PICU

✓ Good Response → Observe for 4 Hours

→ ✓ Improvement → Discharge

→ ✗ No Improvement → Consider Hospital Admission

*In **severe cases**, **heliox** can be considered to reduce respiratory distress in children with severe croup.*

July 2019

Sparsh Shah (MD Student 2020, University of Toronto) for www.pedscases.com

CYSTIC FIBROSIS

CF is an **autosomal recessive** genetic condition characterized by mutations of the **cystic fibrosis transmembrane conductance regulator (CFTR) gene** found on chromosome 7. The most common mutation is **delta F508 deletion**.

PATHOPHYSIOLOGY

Dysfunction of the CFTR protein found in epithelial cells of respiratory, gastrointestinal, reproductive system & sweat glands ⇢ dysregulation of chloride, bicarbonate, & sodium channel transport ⇢ ↓ H2O concentration of secretions ⇢ impaired clearance

EPIDEMIOLOGY

Most prevalent in Caucasians ⇢ ~ 1 in every 3000 – 3500 live births

CLINICAL MANIFESTATIONS

Respiratory

- Progressive disease with intermittent acute exacerbations
- Mucous plugging ⇢ chronic airway inflammation & infections ⇢ bronchiectasis
- **Common pathogens:** S. aureus, H. influenza, P. aeruginosa, B. cepacia

Gastrointestinal

- Intestinal obstruction
- Steatorrhea
- Pancreatic insufficiency, initially exocrine ⇢ eventually also endocrine (CF related diabetes)
- Fat soluble vitamin deficiencies (ADEK)
- CF related liver disease

Other

- Failure to thrive
- Delayed puberty & infertility
- Dehydration & sodium depletion
- Stress incontinence
- Low bone mineral density
- Clubbing

INVESTIGATIONS

- Newborn screening test
- Sweat chloride test
- CF genetic testing

MANAGEMENT

- Airway clearance techniques: chest physiotherapy, nebulized hypertonic saline, dornase alfa
- Regular pulmonary function tests
- Lung transplantation

- Nutrition to maintain BMI >50th percentile
- Pancreatic enzyme supplementation
- Fat soluble vitamin supplementation (ADEK)
- Increased salt intake
- Oral glucose tolerance tests

- CFTR modulator therapies
- Routine immunizations
- Exposure avoidance (no contact with other individuals with CF; no hot tubs or humidifiers)
- Mental health screening (patient & family)

Published February 2021
Vanessa Warren (Medical Student 2021, Northern Ontario School of Medicine) & Dr. Gautam Kumar (General Pediatrician, Northern Ontario School of Medicine) for www.pedscases.com

DEHYDRATION & FLUID REPLACEMENT

Clinical Signs of Dehydration

Degree of Dehydration *Infant/Young Child* *Older Child/Adolescent*	Mild 5% 3%	Moderate 10% 6%	Severe ≥15% ≥9%
Heart Rate	Normal	Rapid	Rapid
Blood Pressure	Normal	Normal	Decreased
Urine Output	Mildly Decreased	Markedly Decreased	Anuria
Mucous Membranes	Moist	Tacky	Dry
Fontanelles	Normal	Sunken	Markedly Sunken
Capillary Refill	Normal (<3s)	Normal to Increased (>3s)	Increased (>3s)

Management of Dehydration*

Mild and Moderate

Oral Rehydration

- → **Fluid**: dilute juice or oral rehydration solutions.
- → Continue **age-appropriate diet** as tolerated.
- → Children ≥6mo can receive a 0.15mg/kg dose (max dose 8mg) of **Ondansetron for vomiting**.
- → If ineffective, move to IV hydration.

Severe

Calculating IV Fluids

- → **STEP 1**: Bolus = NS 10-20mL/kg or 5-10mL/kg if worried about myocardial or renal dysfunction.
- → **STEP 2**: Calculate deficit= % dehydrated x weight in kg.
- → **STEP 3**: Calculate maintenance fluids in mL/hr with 4-2-1 rule.
- → **STEP 4:** Calculate Total Fluid Replacement Over 24h = deficit + maintenance – bolus.
- → **STEP 5**: Divide volume over 24 hours.
- → **STEP 6:** Measure ongoing losses (i.e. vomit, diarrhea, sweat etc.) and replace as needed.
- → **STEP 7:** IV Fluid Selection – Typically D5NS +/- 20mEq/L KCl depending on kidney function and electrolytes.

4-2-1 Rule

Up to 10 kg	4mL/kg
10-20 kg	40mL + 2mL/kg above 10 kg
>20 kg	60mL + 1mL/kg above 20 kg

Example

A 18kg child is 9% dehydrated

- ✓ **Bolus**= 20mL/kg x 18kg = 360mL
- ✓ **Deficit** = 0.09 x 18 = 1.62L
- ✓ **Maintenance fluid** = 40mL + 2(8kg) = 56mL/h
- ✓ **24h Total Fluid Replacement Required** = 1620 + (56 x 24) - 360 = 2604mL
- ✓ **Divide over 24 hours** = 2604/24 hr = 108.5 mL/hr
- ✓ **Replace losses** as needed
- ✓ **IV Fluid** = D5NS +/- 20mEq/L KCl

* Refer to different references for fluid management in infants <28 days old

March, 2022
Grace Georgopoulos (Medical Student, University of Alberta) and Dr. Chris Novak (Pediatrician, Alberta Children's Hospital) for www.pedscases.com

DISORDER OF GUT-BRAIN INTERACTION

Disorders of Gut-Brain Interaction (DGBI) are a group of gastrointestinal disorders with normal findings on bloodwork, imaging or endoscopy but share a common set of symptoms. Functional Abdominal Pain is a type of DGBI.

Other DGBI subtypes include:
- Irritable bowel syndrome
- Functional dyspepsia
- Functional constipation
- Abdominal migraine

TYPICAL FEATURES OF DGBI

Abdominal pain
- Localized around umbilicus
- Worsen with emotional stressors

Nonspecific somatic symptoms
- Headaches
- Arthralgias, myalgias
- Nausea or vomiting
- Constipation or diarrhea

Physical exam
- Abdominal tenderness
- Distractible/transient physical examination findings
- Normal perianal examination
- No systemic findings on exam

DGBIs are the most common cause of chronic abdominal pain and may explain >25% of abdominal pain in children!

DIAGNOSIS

☐ DGBIs are **clinical diagnoses**. Refer to the *Rome IV criteria* for each subtype.

☐ **Rule out pathological causes of abdominal pain** in children including inflammatory bowel disease, gastroesophageal reflux disease, infectious colitis, food allergies, celiac disease by screening for **red flags** or abnormal findings on history, physical, or bloodwork!

☐ Investigations should only be performed as appropriate.

Red flags on history: failure to thrive, delayed puberty, fever, hematochezia, hematemesis, vomiting

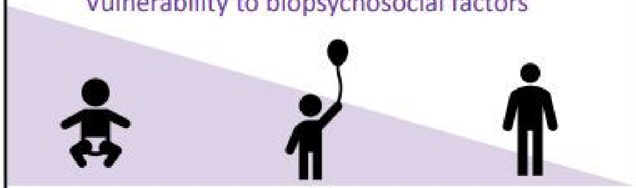
Red flags on physical exam: Uveitis, skin rashes, organomegaly, arthritis, costovertebral angle tenderness, abdominal mass

PATHOPHYSIOLOGY

Genetic predisposition

+

Vulnerability to biopsychosocial factors



Early life trauma
Antibiotic use

Inflammation
Psychological stress

Motility disorders
Coping style

Disordered microbiota gut brain axis

Hypersensitivity to physiological processes occurring within the gut + altered neural signaling of pain

MANAGEMENT

Reassure the patient that their pain experience is real	**Discuss** potential life stressors and work on building insight into pain experience	**Nutrition** counselling to remove triggering foods from diet (track with a food diary)	**Therapy** to cope with stressors and change thinking (CBT)	**Medications** may include laxatives, antispasmodics, antihistamines, antacids, antidepressants, probiotics*

*mixed evidence

June 2024
Anisa Khalfan (Medical Student), Nikhil Pai (Staff Physician, Division of Pediatric Gastroenterology and Nutrition, Michael G. DeGroote School of Medicine) for www.pedscases.com

DOG AND CAT BITES

Key Points:

- **Dog bites** account for **~90%** of animal bites. More likely to cause crush injuries due to powerful jaw strength
- **Cat bites** account for **~10%** of animal bites. More likely to cause infection due to puncture wound with deep inoculum

Investigations:

- **X-ray imaging**
  - Indications: deep bite wounds, including those near joints, crush injuries (dog bites), suspected fracture, suspected foreign body
- **Wound culture**
  - Indication: if signs of infection are present (eg. fever, erythema, swelling, hot to touch, tender, purulence)
- **Blood culture**
  - Indications: If signs of bacteremia are present (e.g. fever, hemodynamic instability, signs of shock) or in immunosuppressed patients

Presentation

History:

- Type of animal
- Time since injury
- Tetanus immunization status of patient
- Details of animal (wild vs. family pet vs. other)
- Signs and symptoms of infection (eg. fever, erythema, swelling, hot to touch, tender, purulence)

Physical Exam:

- Evaluate depth of wound and/ or presence of crush injury (edema, contusion and neurologic dysfunction)
- Look for joint penetration
- Check neurovascular status
- Evaluate for obvious foreign body in wound

⚠ **Deep wounds near vital structures should be treated as penetrating trauma**

Microorganisms

Dog Bite Wound:

- Pasteurella canis
- Capnocytophaga canimorsus**

Organisms Found in Both:

- Pasteurella multocida*
- Bacteroides
- Staphylococcus aureus
- Streptococcus alpha-hemolytic
- Corynebacterium

Cat Bite Wound:

- Bartonella henselae

* Pasteurella multocida is isolated from 75% of cat bites and 50% of dog bites

Management

Wound Care:

- Control bleeding
- Provide local anesthesia if required
- Clean wound with antiseptic solution
- Copious irrigation with sterile saline
- Remove grossly visible debris
- Close wound- weigh risks and benefits of primary intention vs. secondary intention closure

Antibiotics Indications:

- If signs of wound infection or systemic infection are present

Or

- In a clinically uninfected wound with any of the following:
- Deep puncture wounds or lacerations (eg. cat bites)
- Wounds requiring surgical repair or undergoing primary closure
- Wounds on the hand, face or genital area

Rabies and Tetanus Prophylaxis:

- **Rabies:** Perform risk assessment to determine need for rabies post-exposure prophylaxis. Refer to guidelines in your health care region
- **Tetanus:** Determine tetanus immunization status in ALL patients with bite wounds → tetanus prophylaxis administered as indicated

Operative Treatment Indications:

- Crush or devitalized tissue
- Foreign body
- Bites to digital pulp space, nail bed, flexor tendon sheath, deep spaces of the palm
- Septic arthritis
- Abscess formation
- Neurovascular injury
- Functionally or aesthetically sensitive area

July 2023

Emma Yanko (Medical Student, University of Saskatchewan), Dr. John Staples (Pediatric Plastic Surgeon, University of Saskatchewan) for www.pedscases.com

DOWN SYNDROME

- Most common chromosomal condition
- **~1/800 live births**
- Leading genetic cause of intellectual disability
- Every child with Down syndrome is **unique**!

ETIOLOGY

- **Trisomy 21 (94%)**
  - Meiotic nondisjunction
- **Robertsonian translocation (3.3%)**
  - Potentially heritable
- **Mosaicism (2.4%)**
  - Milder phenotype

RISK FACTORS

- Maternal age > 35 years
- Previous pregnancy with chromosomal condition
- Pre-natal screening
  - Nuchal translucency
  - Absent nasal bone
  - Biochemical markers

Diagnosis can be either **pre-natal** (chromosomal karyotype via CVS or amniocentesis) or **post-natal** (chromosomal karyotype if ≥ 2 dysmorphic features seen)

PRESENTATION

CLASSIC FEATURES

- **Craniofacial**
  - Brachycephaly, upslanted palpebral fissures, epicanthal folds, Brushfield spots, midface hypoplasia, flat/short nose, macroglossia, dental hypoplasia, protruding helix, short neck
- **Hands**
  - Single transverse palmar crease, broad fingers, clino/brachydactyly, small 5th mid-phalanx
- **Feet**
  - ↑ space between 1st + 2nd toes
- **General**
  - Hypotonia, hyperflexibility

COMPLICATIONS

- **OPT:** cataracts (✓ red reflex), strabismus
- **HEENT:** hearing loss, recurrent AOM
- **DENT:** periodontal disease
- **CV:** cardiac anomaly (50%)
  - AVSD > VSD, ASD, TOF
- **RESP:** OSA, pulmonary HTN
- **GI:** duodenal atresia, Hirschsprung, Celiac
- **GU:** cryptorchidism, male sterility
- **MSK:** C1/2 instability, hip dysplasia
- **CNS:** development delay, ASD, early Alzheimer's
- **ENDO:** hypothyroid, obesity, short stature
- **HEME:** leukemia (AML, ALL, transient)

MANAGEMENT

SURVEILLANCE

- **GENERAL:** T21 specific growth charts, routine immunizations + RSV (1st yr of life)
- **OPT:** newborn test by 3mo, then yearly
- **HEENT:** newborn hearing screen + behavioral test q6mo until 3yo, then yearly
- **DENT:** consult by 2yo, then every 6mo
- **CV:** newborn echo + peds cardio consult
- **RESP:** OSA monitoring, PSG by 4yo
- **GI:** constipation Tx, Celiac screen at 2yo
- **MSK:** observe for spinal cord compression
- **ENDO:** newborn TSH/T4, then yearly
- **HEME:** newborn CBCd, then CBC yearly

HOLISTIC CARE

- Early lactation support
- Referral to allied health services by 3yo
  - PT/OT/SLP
- Consult teachers to support individualized learning needs
- Mental health support
- Parental support
  - Community resources
  - Advocacy groups
  - Financial assistance

Use Appropriate Language

1. Down *not* "Down's"
2. Use person-first language
3. Be positive
4. End the R-word

April 2022

Summer Hudson (Medical Student, University of Alberta) & Dr. Melanie Lewis (Pediatrician, Edmonton Down Syndrome Clinic, Stollery Children's Hospital) for www.pedscases.com

DUODENAL ATRESIA

Duodenal atresia (DA) is a congenital malformation causing **luminal stricture** of the duodenum, leading to **intestinal obstruction**

Prevalence:
~1 per 10,000 births

Pathophysiology:
Failure of recanalization

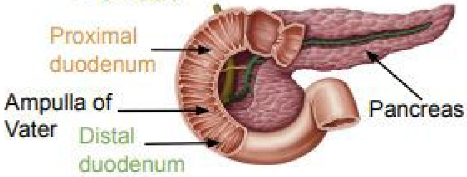
Differential Diagnosis

- Duodenal stenosis
- Antral or pyloric web
- Distal intestinal atresia (e.g. jejunal, ileal, colonic)
- Sepsis
- Annular pancreas
- Meconium ileus
- Malrotation with midgut volvulus

NEONATAL PRESENTATION

HISTORY

- ❑ Vomiting/intolerance of feeds beginning on first day of life
- ❑ Nature of emesis
  - Obstruction proximal to ampulla = non-bilious
  - Obstruction distal to ampulla = bilious



PHYSICAL EXAM

- ❑ Abdominal distension
- ❑ Dehydration
  - Tachycardia, oliguria
  - Sunken fontanelle, dry mucous membranes, poor skin turgor
- ❑ Difficulty breathing (due to aspiration or massive abdominal distension)
- ❑ Jaundice

Important to rule out:
malrotation with midgut volvulus, which requires *immediate* surgical management to prevent midgut infarction

Associated congenital anomalies

- Down Syndrome (Trisomy 21)
  - ~30% of DA patients have T21, but only ~2.5% of T21 patients have DA
- Cardiac, genitourinary, and other chromosomal anomalies

DIAGNOSIS

Prenatal Ultrasound in 2nd or 3rd trimester - Signs of obstruction

- ❑ Two fluid-filled bubbles in stomach and duodenum
- ❑ Polyhydramnios

After Birth

- ❑ **Abdominal Plain-Film X-Ray**
  - ❑ Classic "double bubble" sign with two air-filled bubbles in the stomach and duodenum (Fig. 1)
- ❑ **Upper GI Contrast Study**
  - ❑ Distinguish between distal DA and midgut volvulus

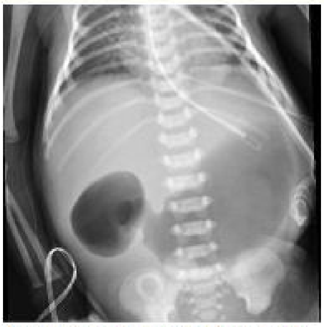


Figure 1. Duodenal atresia

Workup for other anomalies: echocardiogram, renal U/S, rectal biopsy, genetic testing, etc

MANAGEMENT

Immediate management (neonatal unit)

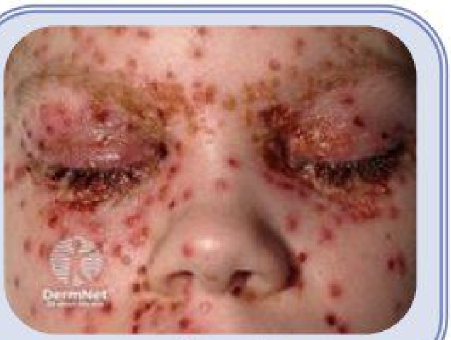
- ❑ NPO, NG tube decompression
- ❑ IV fluid, IV parenteral nutrition
- ❑ Consult pediatric surgery

Operative repair:

- ❑ Anastomosis of functional intestine proximal and distal to the obstruction
  - ❑ In duodenal atresia: duodenoduodenostomy

September 2024
Ariel Qi (Medical Student, Queen's University), Dr. Robert Connelly, (Neonatologist, Queen's University),
Dr. Paula Troncoso (Pediatric Gastroenterologist, Queen's University) for www.pedscases.com

ECZEMA HERPETICUM



source: dermnetz.org

PATHOPHYSIOLOGY

Pathogen:	**Herpes Simplex Virus** (**HSV**) 1 or 2
Cause:	Primary (most common) or recurrent infection
Onset:	2-12 days after exposure
Ages:	All ages, most common in infants and children with **atopic dermatitis** (**AD**)
Risk Factors:	AD patients susceptible due to an impaired skin barrier, inflammation and immune dysregulation [*affects 3-6% of patients with AD*]

PRESENTATION

- Onset: cluster of itchy, painful blisters
- **Morphology**: monomorphic papulovesicles +/- "punched out" erosions overlying eczema. Coalescent, crusted, and overlying an erythematous base
- **Location**: commonly on **head**, **neck**, and **upper torso**

Systemic Symptoms:
- Fever, malaise or lymphadenopathy

DIAGNOSIS

- Clinical diagnosis with high index of suspicion in patient with known AD and new onset monomorphic vesicular rash

INVESTIGATIONS *(for definitive diagnosis)*

- Polymerase chain reaction (PCR) *Gold Standard*
- Alternatives: Direct fluorescent antibody (DFA) testing, Viral culture, Tzanck smear

- Consider blood culture if patient systemically unwell or signs of sepsis

Differential Diagnosis: Impetigo, contact dermatitis, varicella or herpes zoster, eczema molluscatum, eczema coxsackium, monkeypox, hand-foot-and-mouth disease, dermatitis herpetiformis

COURSE

Initial Symptoms (**Day 0-3**)
- fever, malaise, irritability, and painful skin lesions

Progression (**Day 3-7**)
- rapid spread and dissemination

Peak Symptoms (**Week 1-2**)
- pain, discomfort, and extensive skin involvement
- period when complications are most likely to arise

Resolution (**Week 2-6**)

COMPLICATIONS

DERMATOLOGIC EMERGENCY

ACUTE
- Eczema exacerbation
- Secondary bacterial superinfection
- **Ophthalmologic**: Keratitis or Keratoconjunctivitis
- **Neurologic**: Bell's palsy, meningitis, encephalitis
- **Systemic dissemination**:
- viremia, bacteremia, fungemia → multi-organ failure

CHRONIC
- Scarring

- Recurrence in up to 50% of AD patients within first year from initial infection

MANAGEMENT

- If clinical suspicion high or known eczema herpeticum → start acyclovir (IV or PO depending on clinical context)
- If 2° bacterial infection → start antibiotics (cover *s. aureus* and *group A strep.*)
- If involvement of ophthalmologic branch of trigeminal nerve → consult ***ophthalmology***
- If severe and/or recurrent disease → consider prophylactic antiviral therapy
- Optimize AD management (bland emollients, hold topical steroids or calcineurin inhibitor until 48-72h after antiviral)

NOTE: Dosing and treatment is variable based on intravenous vs oral, immune status, and extracutaneous involvement

November 2024
Dylan Hollman (Medical Student, University of Alberta), Dr. Luis Fernando Sanchez-Espino (Pediatric Dermatologist, University of Alberta) for www.pedscases.com

ENURESIS: EVALUATION

Enuresis = persistent nocturnal incontinence

- **>2** episodes/week
- Occurs during **sleep**
- Uncontrolled urinary voiding

- In children ≥**5** years old. Also known as "**bedwetting**"
- Occurs when child does not wake up from sleep to void
- Results from a combination of: **genetic factors, maturational delay, excessively deep sleep, reduced bladder capacity, nocturnal polyuria**

1° Primary Enuresis

Never previously achieved period of nighttime dryness. ~80% of nocturnal enuresis cases, high rate of spontaneous resolution

2° Secondary Enuresis

Enuresis developed after ≥**6 consecutive months** of nighttime continence. Often triggered by **stressors** (divorce, birth of sibling, school), sleep disordered breathing, constipation, suboptimal voiding habits.

Goal: rule out presence of underlying medical problem

Such as urinary tract infection, constipation, obstructive sleep apnea, and diabetes insipidus.

PRESENTATION

HISTORY

- **Frequency** (# /night, # /week, timing of episodes)
- **Trend** (improving vs. worsening)
- **Daytime** symptoms and/or **LUTS***
- **Constipation** or fecal incontinence
- Previous period of dryness (>6mths?)
- **Volume** voided (large volumes are indicative of nocturnal polyuria)
- **Fluid intake** (majority in evening?)
- Previous interventions? Were they successful?
- **PMHx** (sleep apnea, diabetes, UTIs, sickle cell, neurologic abnormalities, ADHD, ASD)
- Neurodevelopmental delays? Behavioural or psychologic concerns?
- **FHx** of nocturnal enuresis (what age did parent resolve enuresis?
- **SHx** (recent stressors?)
- **Impact** on child and family
- Review voiding diary if available

PHYSICAL EXAM

- **Genital exam usually normal** in primary enuresis
- **Fever** may indicate UTI
- Abdominal palpation for **stool**
- **Lower back** for stigmata of spinal dysraphism (midline hair tufts, sacral dimple)
- **Lower limb** strength, tone, reflexes, sensation for evidence of neurogenic bladder

INVESTIGATIONS

If **reassuring** presentation…	If **concerning** history and physical exam…	
No routine tests are required	**Urinalysis & Culture** To **rule out** UTI, DM, DKA, DI	**Renal/Bladder Ultrasound** **Only if** otherwise indicated

EPIDEMIOLOGY & NATURAL HISTORY

Daytime bladder control expected **~age 4**

Nighttime bladder control achieved **months-years** after daytime **~age 5 – 7**

Age	Prevalence
5	15%
6	13%
7	10%
8	7%
10	5%
12-14	2-3%
≥15	1-2%

- Common!
- Resolves spontaneously ~15%/year
- Longer persistence = lower probability of spontaneous resolution

2 : 1
Male : Female

20% also have **daytime** symptoms

15% also have **fecal incontinence**

*Lower Urinary Tract Symptoms (LUTS)

Voiding
- Hesitancy
- Weak stream
- Intermittent stream
- Straining
- Incomplete emptying
- Dribbling

Storage
- Urgency
- Frequency ≥8 or ≤3 times/day

- Genital or lower urinary tract pain
- Holding maneuvers

Presence of daytime symptoms and/or LUTS is suggestive of dysfunctional voiding or anatomical abnormalities, thus should be **referred to Urology**

Published January 2022
Sarah Park (Medical Student C2022, University of Alberta), Dr. Peter Metcalfe (Pediatric Urologist, University of Alberta) for www.pedscases.com

FAILURE TO THRIVE

Failure to Thrive is generally defined as weight <3rd percentile or falling across two major percentile lines.
Choose the appropriate growth chart for gender, age, and genetic syndromes.
The most common cause is **inadequate dietary intake**.

APPROACH

HISTORY

Nutritional Hx: 72h dietary recall, feeding environment (who feeds, timing of meals, grazing, distractions), pickiness, introduction of solids and common allergens, breast or formula fed

Symptoms:
- GI: abdominal pain, stool pattern/quality (Bristol chart, mucous or blood), emesis
- Non-GI: floppy or stiff muscles, fatigue, sweating, cough, shortness of breath, headache

Pregnancy Hx: gestational age, maternal disease, infections

PMHx: recurrent infections, medications, developmental milestones, prior growth trajectory

FHx: parental heights (calculate mid-parental height for growth potential), allergy, genetic syndromes, chronic illnesses listed under "Increased Metabolic Demand"

SocHx: caregiver stressors, mental health, parental expectations

PHYSICAL EXAM

Measurements:
- Weight, height, head circumference (<2 yrs) trended over time
- Use growth charts designed for specific genetic syndromes (e.g., TSM 21, Turner Syndrome)
- Correct for prematurity until 24 months

Focused physical:
- General: dysmorphic features, subcutaneous fat distribution, muscle mass
- Neuro: hypo or hypertonia
- CV, Resp, Abdo: look for signs of chronic disease (e.g., murmur, abdo mass, extra-intestinal manifestations of IBD)
- Caregiver-child interaction
- Developmental/behavioural observation

DIFFERENTIAL DIAGNOSIS

INTAKE ISSUES

- **Insufficient food intake:** difficulty breastfeeding, improper formula mixing, food insecurity, neglect, feeding aversion
- **Mechanical issues:** cleft palate, dental lesions, oromotor issues

INCREASED METABOLIC DEMAND

- Congestive heart disease (CHF)
- **Inflammatory diseases and immunodeficiencies** (e.g., IBD, SLE, celiac disease, cystic fibrosis)
- **Endocrine disorders** (e.g., hyperthyroidism, Type 1 DM)
- **Hematologic issues** (e.g., leukemia)

INCREASED LOSSES

- Vomiting
- Gastroesophageal reflux disease (GERD)
- Malabsorption / Chronic diarrhea

INVESTIGATIONS

- Per CPS guidelines, if clinically indicated after thorough H&P, may obtain basic workup including CBC, renal and liver panels, iron studies, inflammatory markers (ESR, CRP, TTP, IgA), TSH, UA
- Consider karyotype, microarray, bone age (XR of hand and wrist) if also short stature

MANAGEMENT

1. If the patient demonstrates normal growth with adequate caloric intake, no further investigations required.
2. Treat the underlying cause; reassure parents if no underlying disease is detected
3. Provide education about age-appropriate diet, scheduling, child-specific behavioural interventions
4. Fortify food (e.g., increase caloric density – add cream to soup, butter to rice, fortified formula)
5. Refer to lactation consultant, dietician, SLP, or social work as needed

June 2023
Tingting Yan (Medical Student, University of Toronto), Dr. Kim Zhou (Paediatrician, North York General Hospital) for www.pedscases.com

FEBRILE NEUTROPENIA

FEVER: > 38.3°C once OR > 38°C for ≥ 1 hour OR two temperatures > 38°C within 12 hours

+

NEUTROPENIA: Absolute Neutrophil Count (ANC) < 0.5 x 10^9 cells/L

Fever and neutropenia is a **medical emergency**

Febrile neutropenia is a **common complication** in children receiving **chemotherapy** for cancer

ETIOLOGY OF FEVER

- **Bacterial**: bacteremia, line infection, meningitis, pneumonia, UTI, etc.
    - **Gram positive** (most common): *Coagulase-negative Staphylococcus, Staphylococcus aureus, Streptococcus viridans*
    - **Gram negatives**: *E.coli, Klebsiella, Pseudomonas*
- **Viral**: respiratory viruses, *Herpes simplex, Varicella zoster*
- **Fungi**: *Candida, Aspergillus, Cryptococcus*
- **Non-infectious**: drug fever, cancer-related, deep vein thrombosis/ pulmonary embolism, transfusion reaction

There may be no etiology of fever found!

ETIOLOGY OF NEUTROPENIA

- **Highest risk**: chemotherapy
- **High risk**: ill-appearing child with neutropenia (any cause), aplastic anemia, severe congenital neutropenia (ex: Kostmann syndrome)
- **Low-moderate risk**: chronic benign neutropenia, cyclic neutropenia
- **Low risk**: well-appearing transient neutropenia (ex: viral or drug-induced)

HISTORY

Infectious symptoms:

- Site-specific symptoms of **localized infection**
    - Headache, skin changes, oral ulcers, sore throat, cough, SOB, abdominal pain, vomiting, diarrhea, perianal lesions, dysuria, rashes
- **Sick contacts**

Immunization history

Past medical history:

- Underlying diagnosis
- Established central line
- Date of last chemotherapy
- Most recent ANC count
- Previous febrile neutropenia, infections, & antibiotics

Medications & allergies:

- Recent antipyretics
- Antimicrobial prophylaxis and compliance

PHYSICAL EXAM

Careful physical examination to determine site of infection.

Findings may be subtle or none at all!

- **Vitals**: tachycardia & tachypnea often accompany fever
- **Skin**: erythema, temperature, central venous catheter
- **HEENT**: gingivitis, oral ulcers, pharyngitis, cervical lymphadenopathy, meningismus
- **Respiratory**: work of breathing, signs of pneumonia
- **Cardiovascular**: new murmur, cap refill, pulses
- **Abdomen**: tenderness, distension, masses, hepatosplenomegaly
- **Perineum**: perianal and labial lesions

INVESTIGATIONS

INITIAL LABS

- **CBC with differential**
- **Blood cultures** from central line

- Electrolytes
- Urea
- Creatinine

- CRP
- Type & Screen

OTHER CONSIDERATIONS

- +/- urine culture
- +/- swab sites with purulent drainage
- +/- stool cultures

- +/- chest x-ray
- +/- NP swab if respiratory symptoms present

MANAGEMENT ALGORITHM FOR FEBRILE NEUTROPENIA IN CANCER PATIENTS

Initial investigations →

EMPIRIC ANTIBIOTICS
(Goal: 1st dose within 60 minutes of arrival) →

BROAD SPECTRUM ANTIBIOTICS
(e.g: piperacillin-tazobactam & tobramycin)*

→ **Transfer to tertiary care center** for further management by Pediatric Hematology/Oncology.

Recognizing a child who is UNWELL / HEMODYNAMICALLY UNSTABLE:

Early symptoms of shock: lethargy/irritability, poor feeding, fever, ↓ urine output.
Early signs of shock: ↑ HR, ↑ RR ↓ BP, orthostatic BP/pulse changes, delayed cap refill, ↓ level of consciousness. ***Early recognition is critical!***

*Antibiotic treatment is unique to each site based on local sensitivities.

- ➢ Inpatient treatment generally involves **empiric antibiotics** until the **ANC has recovered** and the **fever subsides**. If the ANC is not improving or the fever persists, a **fungal/extended fever workup** is initiated. An **antifungal agent** may be added.
- ➢ For **non-cancer neutropenia**, management is determined by **level of risk**. Consider Pediatric or Pediatric Hematology consultation.
- ➢ The recurrence of fever in a neutropenic host should be approached as a *new fever* and be re-evaluated.

Published October 11, 2020

Katharine V. Jensen (Medical Student 2021, University of Alberta), Dr. Chris Novak (Pediatric Resident, University of Alberta), & Dr. Bev Wilson (Professor and Pediatric Oncologist, University of Alberta) for www.pedscases.com

FEBRILE SEIZURES

A **seizure** occurs when there is a sudden **change in behavior** or **sensation** caused by **abnormal** and **excessive electrical hypersynchronization** of neuronal networks in the cerebral cortex. Normal inhibition is overcome by excessive excitatory stimuli.

FEBRILE SEIZURE: A convulsion, within 24 hours, of a **temp ≥ 38°C** in a child **6 months** to **5 years**, with no previous afebrile seizure history, in the **absence of CNS infection**, **inflammation**, **acute metabolic abnormalities**, etc.

SIMPLE FEBRILE SEIZURES (85%)	COMPLEX FEBRILE SEIZURES	FEBRILE STATUS EPILEPTICUS
➢ Only 1 seizure in a 24-hour period ➢ Generalized; no focal features ➢ Lasting < 15 minutes	➢ Frequent (recurrent seizures in 24 hours) ➢ Focal features ➢ Lasting ≥ 15 minutes	➢ Prolonged febrile seizure ➢ Lasting ≥ 30 minutes
Febrile seizures occur in **2-5%** of children under 5 years old. They are the **most common** type of convulsions.		

HISTORY

- **Seizure history:** pre-ictal, ictal, and post-ictal. Ask about duration, focal symptoms, and provoking events. Do they take an AED?
- **Illness symptoms:** fever, nausea, vomiting, diarrhea, or rash. Symptoms suggestive of otitis media, respiratory illness, or UTI.
- **Past medical history:** neonatal history, perinatal complications, history of seizure, developmental delay, or head injury.
- **Family history:** family history of seizures, including febrile seizures and epilepsy.
- **Medication history**: meds that may lower seizure threshold, including antibiotics (eg: penicillins, metronidazole), anti-asthmatics (eg: theophylline), antidepressants (eg: bupropion, tricyclics), hormones (prednisone, insulin), etc.
- **Other risk factors:** recent immunizations, daycare attendance, developmental delay, etc.

↓

PHYSICAL EXAM

- **ABCs, vitals, level of consciousness**
- **General physical exam:** source of infection (eg: ears, upper respiratory tract, lungs, GI tract, urinary tract, and skin).
- Thorough **neurological exam**
- **Developmental exam**
- **Meningeal signs:**
  - ❑ **Brudzinski's sign**: when the neck is flexed, severe neck stiffness causes a patient to flex their hips and knees.
  - ❑ **Kerning's sign:** flex the thigh at the hip with the knee at 90º, then extend the knee. It is a positive sign if it is painful leading to resistance.
  - ❑ These signs are not present in young infants.

↓

Further diagnostic investigations are **unnecessary** in children with a **typical history** of a **simple febrile seizure** and a **normal neurological exam**.

↓

ACUTE TREATMENT

- IV **benzodiazepines** (lorazepam, diazepam, midazolam) if the seizure has not stopped at 5 minutes.
- **Treat the cause** (eg: infection, metabolic disorders, stop the offending med, etc).

PROPHYLACTIC TREATMENT

- Prophylactic medication is rarely indicated.
- Antipyretics have not shown to prevent recurrence.
- SL/PO **lorazepam** or **diazepam** prescription for children with a history of prolonged febrile seizures (> 5 mins).

↓

EDUCATION / REASSURANCE OF CAREGIVERS

- Febrile seizures are **common**, occurring in **2-5%** of children **≤ 5** years old.
- **No risk of death, brain damage, learning problems,** or **decreased IQ.**
- Most patients have 2 or 3 febrile seizures in a lifetime.

RED FLAGS

Consider **lumbar puncture** if:

- Altered level of consciousness
- Lethargy, irritability
- Meningismus – positive Kerning's or Brudzinski's sign
- Bulging fontanelle – ↑ ICP
- Focal neurological findings
- Age: < 6 months or > 6 years

A CT scan of the head is obtained to establish the safety of performing an LP.

NEUROIMAGING

Indicated in children with:

- Abnormal neurologic examination
- Macrocephaly
- Signs and symptoms of increased ICP (headache, nausea, vomiting, hypertension, confusion, double vision, papilledema)

INVESTIGATIONS

History of **vomiting, diarrhea**, and **decreased fluid intake** or physical exam findings of **dehydration**:

- CBCdiff
- Electrolytes
- Glucose
- Calcium
- Urea

RISK FACTORS FOR RECURRENCE

Recurrence risk: ***40%***

- 1st febrile seizure < 18 months
- Duration of fever < 24 hours
- Complex febrile seizure
- Family history of febrile seizures
- Temperature < 40°C (104°F)

GENERALIZED EPILEPSY WITH FEBRILE SEIZURE PLUS (GEFS +)

- Initially, these children have febrile seizures, but soon develop non-febrile seizures.
- **Treatment**: valproic acid

May 2020
Katharine V. Jensen (Medical Student 2021, University of Alberta) &
Dr. Natarie Liu (Neurologist and Epileptologist, University of Alberta) for www.pedscases.com

FLEXIBLE PES PLANUS

Pes planus or **flat feet**, is a condition where the **arches of the feet are flattened**. Loss of the medial longitudinal arch of the foot, heal valgus deformity, and medial talar prominence.

PRESENTATION

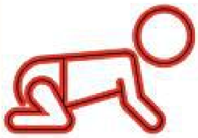
- Generally **asymptomatic** in children
- **Pain** and **discomfort** in bilateral arches, heels, lower legs, feet or ankles
- **Cramps at night**
- **Difficulty standing** on tiptoes
- Foot **fatigue**
- **Swelling**

PHYSICAL EXAM

- **Observation** – in standing, **calcaneal valgus** is apparent, the medial arch will appear **dropped** and there will be **foot eversion**
- **Assess arch height** when sitting, standing, and on tiptoes
- Arch should be present on tip toes if not, rigid pes planus → requires further investigations & surgical referral
- Foot **shape**
- **Range of motion** of foot and ankle
- Joint **stability**
- Tendon & muscle **strength**
- **Gait** & **footwear** assessment

CAUSE

- **Genetic factors** leading to insufficient arch development
- **Injuries** to the foot or ankle
- **Weakness** or **dysfunction** in muscles and tendons supporting the arch



Infants have flexible flat feet with arch development observed around 3 years. It takes until 7 to 10 years for the arch height to reach adult values.

DIAGNOSIS

CLINICAL DIAGNOSIS:

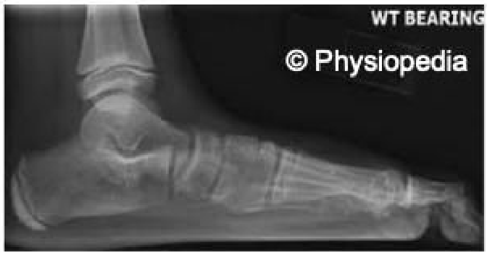
Generally, investigations are unnecessary. **X-rays** can be considered for **severe pain** or concern regarding **bone alignment** or other **structural abnormalities**

TYPES

- **Flexible flat feet:** arches appear when not weight-bearing
- **Rigid flat feet**: arches remain flat regardless of weight-bearing

CO-MORBIDITIES

- Cerebral palsy
- Trisomy 21
- Marfan Syndrome
- Ehlers-Danlos
- Charcot joint
- Tibialis posterior dysfunction
- Obesity
- Arthropathies



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MANAGEMENT

- **Orthotic devices**, such as over the counter arch supports or custom insoles
- Appropriate and supportive **footwear**
- **Physical therapy** exercises to strengthen foot muscles
- Maintaining a **healthy weight** to reduce stress on the feet

August 2024
Dr. Katharine V. Jensen (Pediatrics Resident, University of Alberta) and Dr. Jeanine McColl (Pediatric Rheumatologist, University of Calgary) for www.pedscases.com

FOOD PROTEIN-INDUCED ENTEROCOLITIS

Food protein-induced enterocolitis (FPIES): non-IgE mediated food allergy

Epidemiology:
- Most commonly presents at 2-7 months of age
- Incidence ~0.34%

Pathophysiology: poorly understood, though likely caused by T-cell mediated inflammation

Common triggers

Specific foods	Rates
Cow's milk	67%
Soy	41%
Grains (rice, oat, wheat)	25%
Egg	11%

DIAGNOSIS

FPIES is a **clinical** diagnosis
- Cannot use allergy tests (skin prick, serum IgE)
- Consider oral food challenge (OFC) by an allergist if trigger food unclear, atypical symptoms or persistent symptoms after elimination of trigger food

PRESENTATION

History

- Profuse vomiting, usually 1-4 hours after ingesting the trigger food
- Diarrhea, usually 5-10 hours later
- Irritability
- Lack of cutaneous or respiratory symptoms
- Continued exposure to trigger food may result in abdominal distention, bloody diarrhea, anemia, and failure to thrive

Signs of severe dehydration:
- Rapid heart rate, decreased blood pressure, decreased urine output, dry mucous membranes, sunken fontanelles, increased capillary refill

DIFFERENTIAL DIAGNOSIS

Non-IgE mediated allergies:
- **Food protein-induced allergic proctocolitis**: bloody stools, in an otherwise healthy and thriving infant. Most commonly due to cow's milk and/or soy.
- **Food protein-induced enteropathy** (ex. Celiac disease): diarrhea, vomiting, poor weight gain, abdominal distention, malabsorption

Other:
- **Anaphylaxis**: rapid-onset, serious, multi-system reaction
- **Infections** (ex. gastroenteritis): presence of fever, sick contacts

MANAGEMENT

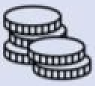
Short-term	Long-term	Prognosis
Treat dehydration: • IV fluid bolus (10-20mL/kg of NS) • Anti-emetics If severe: • In severe presentations, IV corticosteroids may be considered, though evidence is limited 	• Eliminate trigger food from diet • If the trigger was cow's milk, also eliminate soy due to cross-reactivity, and consider replacing with extensively hydrolyzed formula • In most cases, maternal elimination of food is not required while breastfeeding • No epinephrine autoinjector required	• High spontaneous rate of resolution by 3-5 years old • Medically supervised OFCs may be considered as early as 12 to 18 months after the most recent reaction to determine resolution

March 2024

Nikita Menon (Medical Student 2024, UBC), Dr. Odion Kalaci (Pediatrician, Department of Pediatrics, UBC)
for www.pedscases.com

FOREIGN BODY INGESTION

- Foreign body ingestions (FBIs) occur across all ages
  - 75% occur at ≤ 5 years
  - Most common between **6 months – 3 years**
- 98% are **accidental**
- Most commonly **unwitnessed**
- Most common FBs include:
  - **Coins**
  - Button batteries
  - Food boluses
  - Magnets
  - Toys



High Risk Foreign Bodies

- **Button batteries**
  - Can cause mucosal burn + erosion into surrounding structures (e.g. aortoesophageal fistula → life-threatening hemorrhage, esophageal stricture, tracheoesophageal fistula)
  - Continue to cause injury even after removed
- **Multiple magnets (OR single magnet + metallic object)**
  - Attraction across bowel loops → erosion + perforation
- **Large objects (> 6cm x >2.5cm)**
  - High risk of pyloric, duodenal, or ileocecal entrapment
- **Toxic objects**
  - May cause systemic toxicity (e.g. lead)

PRESENTATION

- **Esophageal FB**
  - Drooling
  - Coughing
  - Feeding refusal
  - Dysphagia
  - Neck/chest pain
  - Irritability
  - Bleeding

Hemodynamic instability → aortoesophageal fistula or perforation

- **Gastric/Intestinal FB**
  - Abdominal pain
  - Vomiting
  - GI bleeding

HISTORY

- Witnessed or unwitnessed
- Clarify object, size, ingestion timing
- If food bolus → ask about Hx of atopy + EoE Sx (dysphagia, food sticking)
- Last meal/current NPO status

PHYSICAL EXAM

- First ensure airway stability → **consider FB inhalation**
  - Stridor, tachypnea, work of breathing
- Excessive drooling, oral abrasions/lacerations
- Abdominal tenderness +/- signs of obstruction/peritonitis

DIAGNOSIS

Chest XR → determine type, size, anatomical location of FB

- ☐ **2 views** (AP + lateral) to distinguish coin vs button battery
  - ☐ **Halo Sign:** visible outer ring on AP view → button battery
  - ☐ **Step-Off Sign:** "step" on lateral view → button battery
- ☐ Must include neck to r/o oropharyngeal FB
- ☐ Only radio-opaque FBs visible!

Abdominal XR → as above

Must still XR, even if witnessed ingestion, as child may have ingested another FB before

MANAGEMENT

Button Batteries

- Call Pediatric GI + reference provincial BB guideline
- Honey to prevent mucosal ulceration en route
- **Esophageal** → STAT endoscopic removal
- **Gastric/SB** → need to remove depends on age, Sx, size

Other High Risk FBs

- e.g. Esophageal Coins
- **Esophageal/Gastric** → urgent endoscopic removal
- **Intestinal** → joint decision by Pediatric GI + Surgery

Sharp objects rarely penetrate the GI tract → may not be not considered high risk

Low Risk FBs

- e.g. Gastric Coins
- Discharge w/ guidance to monitor for passage IF:
  - Looks well
  - Pain-free
  - No respiratory distress
  - Tolerates PO intake
- Consider repeat XR in 2-4wks if FB has not passed in stool

May 2022
Summer Hudson (Medical Student, University of Alberta), Dr. Hien Huynh (Pediatric Gastroenterologist, University of Alberta), & Dr. Alex Hudson (Pediatric GI Fellow, University of Alberta) for www.pedscases.com

FRAGILE X SYNDROME

X – linked dominant

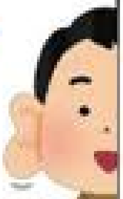
CGG repeat expansion

A genetic disorder caused by a CGG repeat expansion in the FMR1 gene, leading to a lack of FMR protein and abnormal synapse development. It is the **most common inherited cause of intellectual disability**, primarily affecting males, with X-linked dominant inheritance and a prevalence of 1 in 4000.

CLINICAL FEATURES

Growth:
- Macrocephaly
- Accelerated linear growth in early childhood

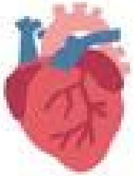
Craniofacial:
- Large protruding ears
- Prominent forehead
- Prominent lower jaw
- Long narrow face
- Strabismus



Psychiatric:
- Behavioral issues
- Social avoidance
- Poor impulse control
- ADHD
- Anxiety
- Depression
- Sleep disorder

Learning challenges:
- Intellectual disability
- Speech delay

Connective tissues:
- Pectus excavatum
- Joint laxity
- Scoliosis
- Mitral valve prolapse



Genitourinary:
- Macro-orchidism
- Dilated ureters
- Vesicoureteral reflux

Gastrointestinal:
- Constipation
- Gastroesophageal reflux disease

DIAGNOSIS

Identification of key clinical features

Confirmatory Genetic Testing:
- FMR1 molecular genetic testing
- CGG allele repeat sizes

- **Normal:** 5 to 44 repeats
- **Intermediate:** 45 to 54 repeats
- **Premutation:** 55 to 200 repeats
- **Full mutation:** 200+ repeats

Fragile X–associated tremor ataxia syndrome:
- Late onset progressive cerebellar ataxia in **premutation-range males**

MANAGEMENT

Behavioral & Educational Support	Medication	Developmental Interventions	Medical Management
Behavioral therapy Special education tailored to **learning needs** **Speech & occupational therapy** for communication and motor skills	*May include:* **Stimulants** for ADHD **SSRIs** for anxiety and mood **Antipsychotics** for aggression	**Early intervention** to address developmental delays **Physical & sensory therapy** to improve motor skills and sensory processing	**Monitor** for seizures, sleep disorders, and connective tissue problems **Manage** gastro-intestinal issues (e.g., constipation, GERD)

Family Support: genetic counseling for families and respite care for caregivers

February 2025

Dr. Katharine V. Jensen (Pediatric Resident, University of Alberta) and Dr. Karen Forbes (Professor of Pediatrics, University of Alberta) for www.pedscases.com

GLOBAL DEVELOPMENTAL DELAY

Global Developmental Delay (GDD) = when children **< 5 years old** experience a **significant delay** in at least **2 domains** of **development**

Developmental Domains:
- Gross Motor
- Fine Motor
- Speech/Language
- Cognitive
- Social

Children with **GDD** often go on to meet the criteria for **intellectual disability**

INITIAL EVALUATION

- ☐ History and physical examination
- ☐ Audiology referral – if concerns in speech/language domain
- ☐ Ophthalmology/Optometry referral – deficits will guide management
- ☐ EEG – if suspect seizures on history

PRESENTATION

HISTORY

- **Developmental Milestones:** parental concerns, milestones met in each domain, regression
- **Birth:** prematurity, growth parameters, NICU – length of stay, complications, APGAR
- **Prenatal:** screening results, antenatal care, maternal medications, substance use, diabetes, HTN, infection
- **Family:** infant deaths, birth defects, neuro + genetic conditions, GDD, neurodevelopmental disability
- **Psychosocial:** abuse, neglect, parental substance use, involvement of children's services

PHYSICAL EXAM

- **Growth Parameters:** height, weight, head circumference (micro or macrocephaly)
- **Appearance:** dysmorphic features
- **Skin:** cutaneous stigmata
- **Head:** shape, fontanelles
- **Cardio:** murmurs, other abnormalities
- **GI:** hepatosplenomegaly
- **GU:** genital abnormalities
- **MSK:** spinal + limb abnormalities
- **Neuro:** tone, strength, reflexes, persistence or absence of primitive reflexes relative to age

ADDITIONAL INVESTIGATIONS

- CBC, AST, ALT, glucose, urea – basic lab work
- Blood gases, electrolytes – to evaluate for acidosis, anion gap
- TSH – hypothyroidism; creatinine kinase – for muscular dystrophy
- Ammonia, lactate, homocysteine, carnitine – for metabolic testing
- Chromosomal microarray, fragile x testing – for genetic testing
- Brain MRI – if abnormal neuro exam, seizures, micro/macrocephaly

CAUSES

Genetic Causes	Chromosomal disorders (e.g. trisomies), single-gene disorders (e.g. Fragile X, Rett syndrome) Inborn errors of metabolism
Prenatal Causes	Toxins/Teratogens – alcohol, lead, radiation Congenital (STORCH) infections
Perinatal Causes	Asphyxia, hypoxia, preterm birth Neonatal complications (infection, trauma, neonatal stroke)
Postnatal Causes	Neglect/malnutrition, infections, trauma, toxins

MANAGEMENT

- Referral to subspecialist – developmental pediatrician, neurologist, genetics
- Early interventions – OT, PT, hearing, vision, SLP, social work, functional living skills
- Family support + education
- Management of underlying cause + comorbidities if applicable/if treatment available

June 2022

Dirusha Moodley (Medical Student, Western University), Dr. Hema Gangam (Pediatric Neurologist, Western University) for www.pedscases.com

HAND, FOOT AND MOUTH DISEASE

Hand, foot and mouth disease (HFMD) is a common viral infection presenting as painful mouth sores and rashes on hands and/or feet. It mostly affects children <7 years old, but can occur in all ages. It is usually a self-limiting illness, but highly contagious occurring from spring to fall in North America.

PRESENTATION

Symptoms may be more severe in infants and young children:

- **Painful oral lesions**
  - on tongue and buccal mucosa
  - may occasionally be absent
- **Maculopapular or papulovesicular rashes** (fluid-filled blisters)
  - on palms (most common) or soles
  - can occur on knees, elbows, buttocks and/or genital area

12-36hr prodrome

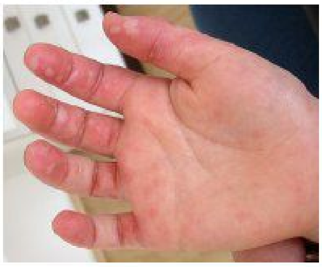
- Fever
- Malaise
- Cough
- Sore throat
- ↓ Appetite
- Abdominal pain
- Myalgia

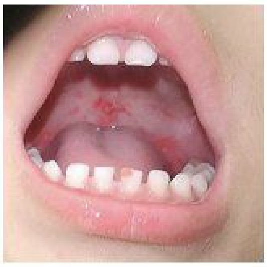
PATHOGEN & TRANSMISSION

- HFMD is caused by Human Enteroviruses, commonly Coxsackievirus A16, A6, Enterovirus 71.
- Infection spread by exposure to virus-containing respiratory droplets, oral secretions, blister fluid or stool. **Enteroviruses may survive on dry surface for several days**.
- Infected persons are most contagious in the first week of illness but virus may be shed in stool for up to 4-8 weeks after symptoms have resolved.

DIAGNOSIS

- HFMD is a **clinical diagnosis**
- Testing for virus is not required for confirmation
- **DDx of vesicular rash:**
  HSV gingivostomatitis (anterior oral/lip lesions), chickenpox (generalized blisters of different ages), Behçet syndrome (recurring oral/genital ulcers).





PREVENTION

- **Hand hygiene**, esp. after changing diapers
- **Disinfect all surfaces**, toys and contaminated items with chlorine bleach and water mixture (ratio: 1 tablespoon bleach to 4 cups water)
- Avoid close contact with infected persons
- Keep child from group settings; talk to daycare and school staff before returning

MANAGEMENT

Prognosis

- Most cases resolve spontaneously in 5-10 days
- Rare neurological/cardiopulmonary complications reported in EV 71 outbreaks in Asia

Treatment

- No effective antiviral therapy or vaccine is available for HFMD
- Supportive treatment of fever, pain and hydration

✓ Give **acetaminophen or ibuprofen** if child is achy or irritable
✓ Try **smaller sips** to stay hydrated
✓ Offer **cold food** if it hurts to swallow

- **Do not** pop the blisters. Keep the areas clean and uncovered
- **Do not** routinely use topical therapies for oral ulcers
- **Never** give aspirin to children or teens
- Educate parents to seek medical treatment if their child has any **sign of dehydration**

June 2024
Annabelle Wong (Medical Student, University of Calgary) & Dr. Bonita Lee (Professor, Division of Pediatric Infectious Disease, University of Alberta) for www.pedscases.com

HEADACHE RED FLAGS

The first-step in managing pediatric headaches is to identify whether it is the emergence of a **Primary Headache** disorder (ex. migraine, cluster, or tension-type headaches) versus a **Secondary Headache** 🚩 caused by an identifiable underlying condition. The SNOOP4 mnemonic is a helpful framework for recognizing important secondary causes of headache.

	QUESTIONS?	SECONDARY CAUSES
Systemic	Fever, night sweats, myalgias, weight loss, chills	Infection, immunocompromise, malignancy
Neurological	Focal or global neurologic deficits: behaviour change, diplopia, visual disturbance, pulsatile tinnitus, seizure, gait disturbance	Neoplastic, inflammatory, infectious, vascular CNS disease
Onset, sudden	Thunderclap headache: How quickly did your pain go from 1/10 to 10/10?	Vascular crises: Stroke, subarachnoid hemorrhage, cerebral venous sinus thrombosis
Onset age <5	While primary headache disorders can present before age 5, new headache under this age is more often secondary in etiology	Neoplastic, inflammatory, infectious CNS disease
Pattern Change **Progressive Headache**	Worsening headache with loss of headache-free periods	Any secondary cause
Precipitated by the Valsalva maneuver	Requires imaging to rule out a secondary cause	Chiari malformation, structural obstruction of cerebrospinal fluid flow
Postural Aggravation	Worse standing up	Intracranial hypotension (CSF leak)
	Worse lying down	Intracranial hypertension
Papilledema	Visual disturbance, diplopia, field defects	Increased intracranial pressure (ICP)

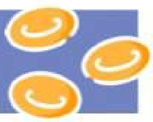
IMPORTANT: If your patient has any **red flag** symptoms, urgent investigations must be performed to rule out a **secondary cause for headache**. ⚠

NEXT STEPS

Imaging: Order CT imaging if you suspect acute hemorrhage or a rapidly enlarging space-occupying lesion. Otherwise, MRI is usually preferred in pediatrics for non-acute situations.
Referral: Urgent referral to pediatric neurology for guidance on further work-up.

June 2024
Julia Craig (Medical Student, University of Alberta), Dr. Thilinie Rajapakse (Pediatric Neurologist, University of Alberta) for www.pedscases.com

HEMOLYTIC ANEMIA



Hemolytic anemia is a group of disorders where red blood cells are destroyed.
Hemolytic anemia can be classified into inherited or immune-mediated causes.
The etiology of hemolytic anemia is relevant to management.

CLINICAL PRESENTATION

SIGNS AND SYMPTOMS

- Pallor
- Fatigue and or lethargy
- Lack of activity
- Changes in school performance
- Dark urine
- Scleral icterus
- Jaundice
- Fever
- Abdominal or back pain
- Dizziness and confusion

PHYSICAL EXAM

- Vital signs
- Level of activity/consciousness
- May appear fatigued
- Check for pallor

HEENT
- Lymph node exam

Cardiac examination
- Tachycardia
- Early systolic flow murmur

Abdo examination
- Splenomegaly
- Hepatomegaly

ETIOLOGY

Intrinsic causes of hemolytic anemia:
- ☐ Hemoglobinopathies: sickle cell disease, thalassemia and unstable hemoglobin
- ☐ Red cell membrane defects: hereditary spherocytes and elliptocytosis
- ☐ Enzyme deficiencies: G6PD deficiency and pyruvate kinase deficiency

Extrinsic causes of hemolytic anemia:
- ☐ Immune hemolysis:
  - Hemolytic disease of the newborn
  - Warm/cold autoimmune hemolytic anemia
  - Acute or delayed transfusion reaction
- ☐ Drug-induced hemolysis
- ☐ Microangiopathic hemolytic anemia (MAHA)
  - Can have infectious triggers

Examples of MAHA:
- Disseminated intravascular coagulation (DIC), including sepsis with DIC
- Hemolytic uremic syndrome (HUS)
- Thrombotic thrombocytopenic purpura (TTP)

INVESTIGATIONS

If you suspect hemolysis:
- ☐ Hemoglobin
- ☐ Reticulocyte count
  - ☐ High: hemolysis/blood loss
  - ☐ Low: decreased bone marrow response
- ☐ Bilirubin
- ☐ LDH
- ☐ Haptoglobin
- ☐ Urinalysis
- ☐ Peripheral smear
- ☐ Type and screen
- ☐ Renal function: urea, creatinine

To determine cause of hemolysis:
- ☐ DAT
- ☐ CBCd for other cell lines
- ☐ PTT/INR
- ☐ Fibrinogen
- ☐ G6PD def screening
- ☐ Hemoglobinopathy investigation for thalassemia and sickle cell disease
- ☐ EMA flow cytometry +/- osmotic fragility
- ☐ Pyruvate kinase enzyme activity
- ☐ Genetic testing as indicated

Hemolysis Labs

Test	Result
Bilirubin	↑
LDH	↑
Reticulocytes	↑
Haptoglobin*	↓

*Serum haptoglobin is not reliable in infants under 4 months of age.

Hemolytic anemia should be treated as an **emergency** until you know the cause and rate of hemolysis due to the risk of **severe anemia** and **renal failure**.

MANAGEMENT

Acute management of all hemolysis:
- ☐ Stop cause if found (ie. medication)
- ☐ Hydrate at 1.5x maintenance
- ☐ Watch urine output
- ☐ Serial monitoring of hemoglobin
- ☐ Consider pRBC transfusion depending on cause and rate of hemolysis

Management can depend on specific etiology:
- ☐ Warm AIHI: corticosteroids
- ☐ Cold AIHI: supportive care if mild, consider rituximab or exchange transfusion if severe
- ☐ Transfusion reaction: STOP transfusion, supportive care
- ☐ Treat cause of hemolysis (e.g. sepsis)

Published May 2022
Morgan Gregg (Medical Student, University of Alberta), Dr. Corriveau-Bourque (Pediatric Hematologist, University of Alberta) for www.pedscases.com

HIRSCHSPRUNG DISEASE

- Congenital defect
- Incidence: 1:5000 live births
- M:F = 4:1
- 85% of the time limited to the rectosigmoid segment of the colon

PATHOPHYSIOLOGY

Migration failure of neural crest cells to the distal colon

↓

Absence of ganglion cells in the submucosal and myenteric plexuses

↓

Absence of peristalsis in the aganglionic colon

↓

Functional obstruction

CLINICAL PRESENTATION

NEWBORN	OLDER CHILD
• Failure to pass meconium in first 24 hours after birth • Bowel obstruction with bilious vomiting	• Severe chronic constipation • Failure to thrive, malnutrition and feeding problems

PHYSICAL EXAM

- Abdominal distension
- Digital rectal exam:
  - Empty rectum
  - Explosive ejection of stool as the finger is removed ("squirt sign")

INVESTIGATIONS

- TSH (rule out hypothyroidism), electrolytes (rule out hypercalcemia, hypokalemia)
- Abdominal X-Ray → will show dilated loops of intestine
- Contrast enema → will show the "transition zone" between the narrow rectum and distal colon and the dilated proximal colon
  - Contraindicated if enterocolitis is suspected → risk of perforation
  - For infants > 12 months old → anal manometry can show absence of rectoanal inhibitory reflex

DIAGNOSIS:

Rectal biopsy → will show aganglionosis, hypertrophy of nerve trunks, and abnormal calretinin staining

TREATMENT

Surgical "pull-through" procedure: removal of the aganglionic segment and anastomosis between the normally innervated part of the intestine and the anus, preserving the anal sphincter

HIRSCHSPRUNG-ASSOCIATED ENTEROCOLITIS

- Life-threatening complication
- Inflammation of the colon which causes bowel obstruction & sepsis
- Can occur either before or after the pullthrough surgery

Signs and symptoms

- Abdominal distension
- Explosive diarrhea
- Vomiting
- Other: fever, rectal bleeding, shock
- X-ray: megacolon (severely dilated colon)

Treatment

- Broad spectrum antibiotics
- IV fluids
- Labs: CBC, CRP, blood culture
- Nasogastric decompression
- Rectal irrigations
- General surgery consultation

Published May 2021
Shuxia Côté-Sergerie (Medical Student, University of Sherbrooke) and Dr. Jacob Langer (Pediatric Surgeon & Professor of Surgery, University of Toronto) for www.pedscases.com

HYPERTROPHIC PYLORIC STENOSIS

Hypertrophic pyloric stenosis (HPS) is the **most common cause of gastric outlet obstruction** in infancy from **2-12 weeks CGA.** HPS is **rare before 2 weeks** of age in a term infant as **pyloric musculature hypertrophies** until gastric outlet obstruction occurs. This obstruction causes the classic symptoms and lab findings of HPS.

PRESENTATION

- Usually well before symptom onset (median 6 weeks)
- Non-bloody, non-bilious, **projectile** vomiting **after** feeds
- Appears **hungry** post-feed
- Dehydration and weight loss
- Distended abdomen
- Visible **peristaltic waves**

Although rare, if there is a palpable thickened mass in RUQ (the "olive"), this is pathognomonic of HPS.

RISK FACTORS

- 2-5 weeks at symptom onset
- Male gender
- First born
- Formula feeding
- In utero / neonatal macrolide exposure
- Parental history of HPS
- C section delivery
- Certain Genetic syndromes:
  Cornelia de Lanage Syndrome, Smith-Lemli-Opitz Syndrome, Apert Syndrome, Down Syndrome, and Trisomy 18 Syndrome

Differential Dx of Infantile Non-Bilious Emesis

☐ Hypertrophic pyloric stenosis
☐ GER/GERD
☐ Duodenal stenosis (proximal to Ampulla of Vater)
☐ Gastroenteritis
☐ Cow's milk protein intolerance
☐ Inborn errors of metabolism
☐ Liver disease
☐ Gastric, antral, or pyloric atresia
☐ Pyloric or antral membrane
☐ Gastric volvulus
☐ Overfeeding

INVESTIGATIONS

☐ Abdominal ultrasound: positive if muscle thickness >3mm and length ≥15mm
☐ Upper GI study if US unavailable → "string sign"
☐ Observed feeding trial if imaging is inconclusive
☐ Metabolic panel with electrolyte assessment
☐ Bilirubin if jaundiced

PATHOPHYSIOLOGY

☐ Impaired neuronal nitric oxide synthase synthesis may be impaired in HPS → disrupted smooth muscle relaxation in myenteric plexus → pyloric hypertrophy
☐ Gastric hyperacidity may also play a role in causing HPS, although the exact etiology is unknown.

ELECTROLYTE ABNORMALITIES IN HPS
↓ Chloride
↓ Potassium
Metabolic Alkalosis

SIGNS OF HYPOVOLEMIC SHOCK:
Severe dehydration:
☐ Altered mental status
☐ Impaired end-organ perfusion
☐ Decreased blood pressure

ALTHOUGH UNCOMMON IN HPS, RAPID FLUID RESUSITATION IS REQUIRED IF THESE SIGNS ARE PRESENT.

MANAGEMENT

Consult Gen Surg

1. Correct any metabolic or electrolyte abnormalities
2. Assess hydration status and correct fluid deficit
   ☐ Electrolyte imbalance and dehydration must be corrected prior to anesthetic
3. Laparoscopic pyloromyotomy is the standard approach: surgery is only definitive treatment
4. Can resume oral feeds within a few hours of surgery, often with some regurgitation but an excellent long-term prognosis when identified early.

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Morgan Gregg (Medical Student), Dr. Foulds (Pediatrician, University of Alberta) for www.pedscases.com

HYPOTHYROIDISM

DEFINITION: thyroid hormone *deficiency*

Congenital: thyroid gland development defect (dysgenesis) (85%), thyroid hormone synthesis defect (10-15%)

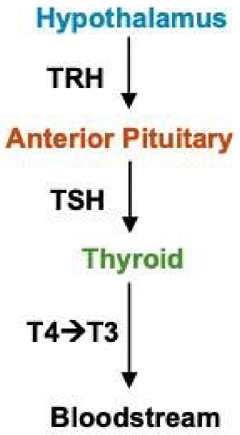
Acquired: autoimmune (Hashimoto's), drugs (antiepileptics, etc), thyroid injury (radiation), iodine deficiency (rare)

More common with family history of autoimmune conditions, Turner Syndrome, and Down's Syndrome

Most common **preventable** cause of intellectual disability

PATHOPHYSIOLOGY

Normal regulation of HPT axis



Dysregulation of HPT axis in hypothyroidism

3° hypothyroidism – hypothalamic dysfunction (↓ TRH)

2° hypothyroidism – pituitary dysfunction (↓/N TSH)

1° hypothyroidism – inflammation/destruction of the thyroid (↑ TSH, ↓T4)

CLINICAL MANIFESTATIONS

Infants (often asymptomatic)

Early signs:
- Prolonged jaundice
- Pallor
- Large anterior fontanelle
- Hypotonia
- Edema
- Hypothermia

Late signs:
- Poor feeding
- Poor growth
- Umbilical hernia
- Developmental delay
- Macroglossia
- Lethargy

Children & Adolescents
- ***Short stature***
- Delayed bone age
- Puberty delay
- Menstrual irregularity
- Goiter
- Fatigue
- Constipation
- Dry Skin

DIAGNOSIS
- **Routine newborn screening** used before clinical signs develop and if positive, confirm with thyroid function tests
- In children, consider ordering a **TSH** if there is clinical suspicion of hypothyroidism

INVESTIGATIONS

Type	TSH	T4	Abnormality
Primary	↑	↓	Thyroid
Secondary/ Tertiary	↓	↓	Pituitary/ Hypothalamus
Subclinical	↑	-	Thyroid

MANAGEMENT

Congenital
- Start Levothyroxine **immediately**
- Confirm etiology with **thyroid ultrasound**
- Monitor TSH frequently

Acquired
- Start Levothyroxine & treat until TSH returns to normal
- Check **antithyroglobulin antibodies**
- Monitor TSH every 6-12 months & after dose change

- **Subclinical hypothyroidism**: continue to monitor and treat if symptoms or a goiter develop

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Gillian Nearing (Medical Student 2023, Northern Ontario School of Medicine) & Dr. Jonathan DellaVedova (General Pediatrician, Northern Ontario School of Medicine) for www.pedscases.com

IMMUNE THROMBOCYTOPENIA (ITP)

- ITP is an **immune-mediated** condition leading to **platelet destruction**
- Affects healthy children over 3 months old, most common in those aged 2 to 5 years
- Typically **self-limited:** resolves in 75%-80% of cases within 6 months and 90% within 1 year
- **Incidence:** 5 per 100,000 children annually
- Etiology unknown, often viral trigger

PRESENTATION

The most serious complication of ITP is **intracranial hemorrhage** (occurs in 0.1% to 0.4% of cases).

PHYSICAL EXAM

- Sudden onset mild petechiae and bruising
- 3% of children have bleeding from nose, mucosa, or gastrointestinal tract
- Typically, well appearing child

DIAGNOSIS

Consider classic features on **history**, **physical examination**, and **laboratory investigation.**

INVESTIGATIONS

Isolated thrombocytopenia: Platelet count <100 x 10^9/L, but most cases have a platelet count of <20 x 10^9/L

Normal blood smear, white blood cell count, hemoglobin

Differential Diagnosis: Malignancy, bone marrow failure, autoimmune disorder, infection

PATHOPHYSIOLOGY

- Antibodies directed against platelets causing substantial decrease in count (↑ destruction)
- **Primary ITP:** Immune-mediated thrombocytopenia; Idiopathic
- **Secondary ITP:** Immune-mediated thrombocytopenia with an underlying cause, such as drug-induced, systemic lupus erythematous, immune deficiencies, malignancy (requires further investigation)

Red Flags on History	Red Flags on Physical Exam	Red Flags on Investigations
Constitutional symptoms Bone pain Poor response to treatment Recurrent thrombocytopenia	Unwell appearing child Lymphadenopathy Hepatomegaly Splenomegaly Signs of chronic illness	Low hemoglobin (no recent explanation of bleeding history) High mean corpuscular volume Abnormal white blood cell or neutrophil count Abnormalities in the blood smear

In children with red flag features, **further investigations are required.**

MANAGEMENT

- Management recommendations depend on severity of symptoms and shared decision making with families
- If not resolved by 6 months, consult hematology

No or mild bleeding (skin manifestations only)	**Moderate bleeding (active mucosal bleeding)**	**Severe bleeding (bleeding requiring hospitalization and/or blood transfusion)**
Outpatient management with observation or short-course of oral corticosteroids	Active therapy with oral corticosteroids or intravenous immunoglobulin (IVIG)	Immediate treatment with corticosteroid, IVIG, and platelet transfusion, depending on severity. Consult hematology.

December 2024

Ajantha Nadarajah (Medical Student, University of Toronto), Dr. Jeremy Friedman (Hospital Pediatrician and Professor, University of Toronto), Dr. Carolyn Beck (Hospital Pediatrician and Associate Professor, University of Toronto) for www.pedscases.com

ELEVATED INTRACRANIAL PRESSURE (ICP)

Signs & Symptoms

	ACUTE	CHRONIC
INFANT	Vomiting, brady/tachycardia, irritability, altered LOC, bulging ant. fontanelle, *Cushing's triad	Bulging anterior fontanelle, irritability, lethargy, poor feeding, vomiting
CHILD (verbal)	Headache, vomiting, altered LOC, papilledema, hypertension, brady/tachycardia, *Cushing's triad	Headache and vomiting (worse in the morning), visual disturbance, abnormal gait, poor coordination, papilledema, Perinaud's syndrome

*Cushing's Triad (late finding): hypertension, bradycardia, irregular respirations

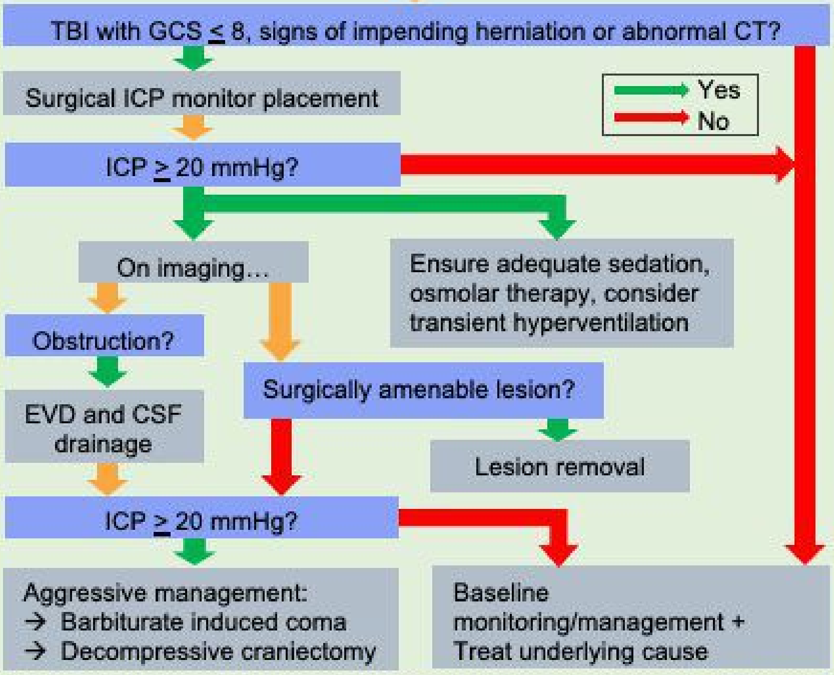
Management of Elevated ICP in Hospital

Symptomatic increased ICP or signs of impending herniation

1. **Baseline Stabilization**

→ Airway, Breathing, Circulation
→ Elevate head to 30° and keep midline
→ Maintain normoxemia and normocarbia (35-40mmHg)
→ Avoid hypotension with PRN fluids, vasopressors
→ Avoid hyperthermia with Acetaminophen or mechanical cooling
→ Sedation for agitation
→ Osmolar therapy (mannitol or hypertonic saline)
→ Intubate if refractory hypoxia, hypoventilation, GCS ≤ 8 or loss of airway protection

2. **Emergent CT**



TBI with GCS ≤ 8, signs of impending herniation or abnormal CT?

Surgical ICP monitor placement

ICP ≥ 20 mmHg?

On imaging…

Ensure adequate sedation, osmolar therapy, consider transient hyperventilation

Obstruction?

EVD and CSF drainage

Surgically amenable lesion?

Lesion removal

ICP ≥ 20 mmHg?

Aggressive management:
→ Barbiturate induced coma
→ Decompressive craniectomy

Baseline monitoring/management + Treat underlying cause

Differential Diagnosis

Reduced intracranial space due to:

1. CSF: hydrocephalus, increased production
2. Blood: hematoma, hemorrhage, vasodilation
3. Brain/skull: mass (causing bleeding), cerebral edema, depressed skull fracture
4. Idiopathic intracranial hypertension

Physical Exam

→ Airway, Breathing, Circulation
→ Vitals
→ Neurologic Exam and GCS

Investigations

→ Labs: Electrolytes (avoid hyponatremia), Ca^{2+}, Mg^{+}, urea nitrogen, Cr, ABG, CBCd, pan-culture (if febrile)
→ Imaging: CT or MRI
→ Procedures: *LP

*LP is **contraindicated** in patients with a known/ suspected intracranial mass or suspicion of highly elevated ICP

Elevated ICP

ICP ≥ 20mmHg for >5min

Suspect Non-Accidental Injury if:

→ **Acute elevated ICP + retinal hemorrhage**, cutaneous bruising, fractures or visceral injury

Management Memory Aid

ICP HEAD
→ **I** Intubate
→ **C** Calm (sedate)/Coma
→ **P** Place drain/Paralysis
→ **H** Hyperventilate
→ **E** Elevate head
→ **A** Adequate BP
→ **D** Diuretic (i.e. mannitol)

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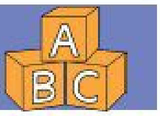
Grace Georgopoulos (Medical Student 2022, University of Alberta) & Dr. Jonathan Duff (Pediatric Intensivist, University of Alberta) for www.pedscases.com

INFANT FEEDING



0 – 6 months **EXCLUSIVE BREASTFEEDING recommended for first 6 months**

- Breastfeeding can continue for **2 years or longer** as desired by mother & infant
- The only supplement breastfed infants require is **vitamin D3 400 units PO daily** (avoid D2 & multivitamins)
- **Spitting up is common** in infants & shouldn't lead to discontinuation of breastfeeding

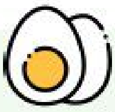
Do:

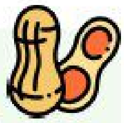
- ✓ Reassure parents that newborns normally experience weight loss in the first 1-2 weeks
- ✓ Should **regain birth weight by 10-14 days of life**, and continue to grow steadily thereafter (~20-30g/day for first 3 months, then 75-200 g/week from months 3-6)
- ✓ Encourage regular check ups. Nutrition monitoring can be done using **WHO Growth Charts**

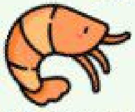
Failure to Thrive (FTT)

Sometimes defined as **weight <3rd percentile** on appropriate growth chart, or when infant **crosses two major percentiles.** The most common cause is **inadequate caloric intake.**

4 – 6 months **INTRODUCTION OF ALLERGENIC FOODS**





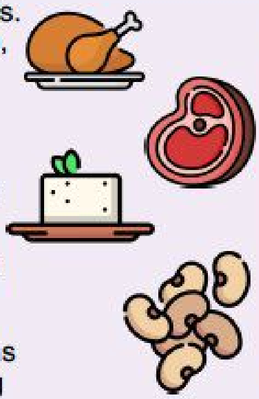


- Early **introduction of allergenic foods between 4-6 months** of age may have a role in preventing food allergy, particularly egg and peanut in high-risk infants
- Allergenic foods: **peanuts, tree nuts, egg, soy, shellfish**, and others
- High-risk infants: those with a 1st degree relative with atopy (eczema, asthma, allergies)
- No more than **1 potential food allergen should be introduced at one time**. Wait 2 days to assess for any signs or symptoms of allergic reaction
- No evidence that the order of solid food introduction affects the risk of food allergy development

6 – 12 months **COMPLIMENTARY FOODS INTRODUCED**

Iron-rich solid foods include: Iron-rich foods. Examples include meats (beef, lamb, poultry), seafood and fish, meat alternatives & iron fortified cereals (tofu & legumes cooked until soft)

- Iron stores from maternal nutrition deplete by ~6 months old
- Complementary feeding should be offered by latest 9 months old
- Exposing children to as many new foods as possible can help promote healthier eating habits later in life



Signs of readiness for complementary foods

- ➢ Infant has better control of their head, can sit up & lean forward
- ➢ Infant can pick up food and put in their mouth, as well as shows signs they're full (ex. turning head away)
- ➢ Infant can express 'interest' in feeding (ex. watch others eat)

9 – 12 months **PASTEURIZED MILK**

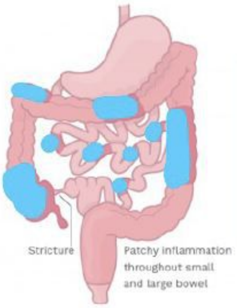
- Pasteurized Homogenized (3.25% M.F.) cow's milk may be introduced

Avoid honey under the age of 12 months old. Honey = only food directly linked to infant botulism, as even pasteurization does not kill the spores of *Clostridium botulinum*.

Published July 2021
Michelle Schneeweiss (Medical Student, McMaster University) and Dr. Jason Silverman (Pediatric Gastroenterologist, University of Alberta) for www.pedscases.com

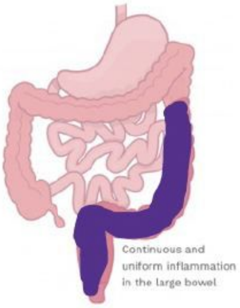
INFLAMMATORY BOWEL DISEASE (IBD)

	Crohn's Disease (CD)	Ulcerative Colitis (UC)
Pathophysiology	In children, CD is **more common** than UC. Inflammation in CD is usually **transmural** and can affect any part of the GI tract ("**gum to bum**")	Inflammation in UC **only affects the intestinal mucosa** of the **colon**. Etiology is a combination of genes, environment, and gut microbiota dysbiosis
Presentation	Abdominal pain, bloody diarrhea, weight loss Can also have RLQ pain (terminal ileitis), **perianal** disease, growth retardation, delayed puberty, short stature, nausea or vomiting, and fatigue	Diarrhea, hematochezia, abdominal pain Can also have tenesmus, urgency. May have acute weight loss



Extra-intestinal manifestations

Seen in both CD and UC! Erythema nodosum (tender erythematous nodules usually on the shins), pyoderma grangrenosum (small, red nodules that erode into painful ulcerations), oral ulcers, arthralgias, arthritis. Primary sclerosing cholangitis (PSC) can happen in CD or UC, as well as autoimmune hepatitis.



Lab Tests
CBC → anemia
ESR, CRP → may be elevated
Hypoalbuminemia
AST, ALP, GGT → can be abnormal
Fecal Calprotectin → elevated
Stool Cultures (to exclude infection)

Investigations

- Both **gastroscopy and colonoscopy** performed (since CD can be anywhere gum to bum).
- MR enterography helps assess severity and distribution.

Differential diagnosis

- ➢ Gastroenteritis
- ➢ Celiac disease
- ➢ Malabsorption syndromes
- ➢ Irritable bowel syndrome (IBS)

MANAGEMENT

Goal is to achieve remission and promote mucosal healing.

CD:	UC
Induction: EEN (exclusive enteral nutrition) or corticosteroids.	**Acute flares**: 5-Aminosalicylates and corticosteroids.
Maintenance: immunosuppression via methotrexate, azathioprine, or biologics. Surgery is not a curative treatment modality.	**Maintenance**: immunosuppressive (sulfasalazine, azathioprine, biologics). May be surgically resectable.

Toxic megacolon can be a complication!! Dx made with AXR/CT. Fluids, anti-inflammatories, and antibiotics for treatment. May need surgery if no improvement on meds.

Published December, 2021
Prachi Shah (MD Student C2023, University of Alberta), Dr. Matthew Carrol (Pediatric Gastroenterologist, Stollery Children's Hospital), for www.pedscases.com

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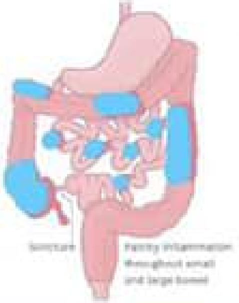

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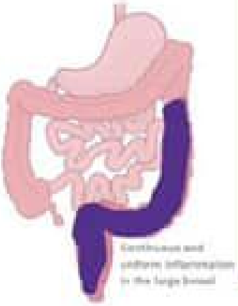

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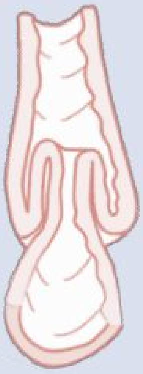
Published December, 2021
Prachi Shah (MD Student C2023, University of Alberta), Dr. Matthew Carrol (Pediatric Gastroenterologist, Stollery Children's Hospital), for www.pedscases.com

INTUSSUSCEPTION

Intussusception is defined as a **telescoping** of a part of the intestine into the lumen of adjoining intestine.

This most often occurs idiopathically; however, a **lead point** may be implicated (e.g. enlarged Peyer's patches or sites of anatomic abnormality)



PRESENTATION

HISTORY

Epidemiology

- ☐ Male > female
- ☐ 3 months to 3 years old (peak 6 to 12 months)
- ☐ Recent viral illness

Clinical Symptoms

- ☐ Episodic abdominal pain
- ☐ Lethargy
- ☐ Vomiting
- ☐ Red "currant jelly" stools (uncommon)
- ☐ Poor feeding

PHYSICAL EXAM

General Exam

- ☐ Tachycardia, hypertension, pallor
- ☐ Patient is uncomfortable & unwell
- ☐ **Late sign:** hypovolemic shock (tachypnea, tachycardia, lethargy)

Abdominal Exam

- ☐ May be normal
- ☐ Palpable abdominal mass
- ☐ Abdominal distension
- ☐ **Late sign:** peritonitis (rigidity, guarding, rebound tenderness)

DIAGNOSIS

Abdominal Ultrasound

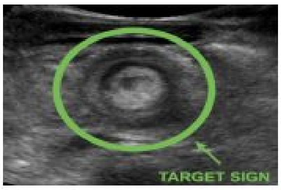
- ☐ Can be used as quick initial diagnostic test if diagnosis is uncertain
- ☐ Look for 3-5cm mass deep to abdominal wall with target sign appearance

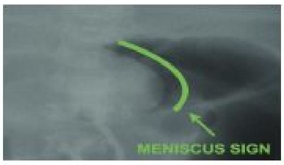
Enema (Liquid Contrast or Air)

- ☐ Most sensitive and specific test that is both diagnostic and therapeutic
- ☐ Look for meniscus sign or filling defect

Abdominal Plain-Film X-Ray

- ☐ Poor sensitivity and specificity for diagnosis of intussusception
- ☐ Useful to rule out suspected obstruction or perforation of bowels





⚠ **CONSULT SURGERY IMMEDIATELY** if suspected peritonitis, shock or free intra-abdominal air
(note: enema is contraindicated in these cases)

MANAGEMENT

- ☐ Fluid resuscitation (isotonic IV)

Enema Reduction

- ☐ Best option for clinically stable patients
- ☐ Enema can be air, saline, water soluble contrast, or ultrasound-guided (note: no clear advantage among enema modalities)

OR

Surgical Reduction

- ☐ Best option for unstable patients (hypovolemic shock, peritonitis)
- ☐ Laparoscopic or open surgery to milk out the intussusception
- ☐ If reduction fails or bowel is non-viable, resection of bowel area is performed

PROGNOSIS

Excellent prognosis if treated early

Late diagnosis can lead to bowel ischemia, sepsis and risk of mortality

Intussusception reoccurs after ~10% of enema reductions, 2-5% of surgical reductions

November 2023
Jonah Isen (Medical Student, Queen's University), Dr. Andrea Winthrop (Pediatric Surgeon, Queen's University) for www.pedscases.com

INVASIVE MENINGOCOCCAL DISEASE

Terminology

Invasive meningococcal disease includes:

1) Meningococcal meningitis: Infection and inflammation of the meninges caused by *Neisseria meningiditis*.

2) Meningococcemia: Infection of the bloodstream caused by *Neisseria meningiditis.*

MAIN RISK FACTORS

- Unimmunized or incompletely immunized individuals
- Primary or secondary immunodeficiency (congenital or acquired, such as asplenia/hyposplenia and auto-immune disease)

INVESTIGATIONS

- **Gold standard**: isolation of N. meningitidis from sterile body fluid (blood culture or CSF)
- CBC, electrolytes, CRP, glucose, creatinine, ALT: leukocytosis, electrolyte abnormalities, acute kidney failure
- Arterial blood gas: metabolic acidosis may be present
- If septic: lactate, PT, PTT, fibrinogen, d-dimers, CK
- Neuroimaging (head CT) if neurologic symptoms are present
- Lumbar puncture: CSF analysis and culture, gram staining, PCR

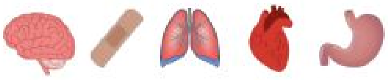
MANAGEMENT

- **Medical emergency** → Early administration of antibiotic therapy (do not delay for lumbar puncture)
- High dose IV Ceftriaxone pending susceptibility testing
- Dexamethasone: reduces neurological sequelae, morbidity & mortality
- Supportive care

CLINICAL PRESENTATION

History

- **Sepsis** and/or septic choc
- **Neurological** (meningitis) : fever, nausea, vomiting, headache, photophobia, difficulty concentrating, myalgias, impaired consciousness
- **Dermatologic**: Petechial rash progressing to purpura fulminans (maculopapular rash progressing to blood filled blisters and eventually leading to infarction-related skin necrosis)
- **Respiratory**: sore throat, coryza
- **Cardiac**: myocarditis, heart failure
- **Abdominal**: abdominal pain and gastroenteritis (acute abdomen)



Physical exam

- **Meningeal irritability**: positive Kernig and/or Brudzinski signs
- Vital sign changes often consistent with **"warm shock":** bounding pulses, very low diastolic blood pressure with wide pulse pressure, tachycardia, diaphoresis
- **Rash**: petechiae 1-2mm in diameter concentrated on trunk & lower portions of the body (can also present as large purpuric and ecchymotic lesions)

COMPLICATIONS

Lack of end organ failure:

- Renal insufficiency
- Adrenal infarction
- Disseminated intravascular coagulation

- Sensorineural hearing loss
- Pleuritis
- Endocarditis
- Amputation

PREVENTION

- Isolation: droplet precautions until 24 hours of appropriate antibiotic therapy
- **Notification to local Public Health department**
- Early antimicrobial chemoprophylaxis in close contacts: Rifampin, Ciprofloxacin or Ceftriaxone based on antimicrobial susceptibility
- Meningococcal conjugate vaccine and/or serogroup B meningococcal vaccine: immunoprophylaxis in previously vaccinated closed contacts should be considered

August 2023
Elizabeth Di Flumeri (Medical Student, University of Sherbrooke) & Dr. Alexandra Langlois (Pediatric immuno-allergist & Professor, University of Sherbrooke) for www.pedscases.com

JUVENILE IDIOPATHIC ARTHRITIS

DIAGNOSIS

1. Arthritis in ≥ 1 joint for ≥ 6 weeks
2. < 16 years old
3. Exclusion of other disease that may cause arthritis

Arthritis → joint pain, stiffness, decreased range of motion, swelling

CLINICAL PRESENTATION

TYPE	TYPICAL AGE	AFFECTED JOINTS	JOINT PATTERN	EXTRA-ARTICULAR FEATURES
Oligoarthritis	< 6y	1 – 4	Asymmetric Large joints	Asymptomatic uveitis
RF- Polyarthritis	6-7y	≥ 5	Symmetric Any sized joints	Asymptomatic uveitis
RF+ Polyarthritis	9-12y	≥ 5	Symmetric Any sized joints	Rheumatoid nodules, uveitis
Systemic Arthritis	2-4y	≥ 1	Any sized joints	Fever, uveitis, lymphadenopathy, rash, serositis, hepatosplenomegaly
Psoriatic Arthritis	7-10y	≥ 1	Asymmetric/symmetric Small-medium joints	Dactylitis, nail pitting, psoriasis, uveitis
Enthesitis Related Arthritis	9-12y	N/A	Tendon insertions Lower extremities	Symptomatic uveitis

INVESTIGATIONS

Diagnosis of JIA is based on clinical findings, investigations are used to rule out other conditions and may help classify type.

- CBC
- ESR/CRP
- RF, ANA
- XR, U/S, MRI, CT

Children presenting with systemic symptoms should be worked up to rule out infection/malignancy.

DDX

- Infection (septic arthritis, osteomyelitis)
- Malignancy (acute leukemia)
- Growing pains
- Trauma
- Inflammatory Bowel Disease
- Juvenile Dermatomyositis
- Systemic Lupus Erythematous
- Vasculitis

MANAGEMENT

PHARMACOLOGIC

- NSAIDS (ibuprofen, naproxen)
- Intra-articular steroids
- Systemic steroids (short course)
- Disease Modifying Anti-Rheumatic Drugs (methotrexate)
- Biologics

NON-PHARMACOLOGIC

- Regular physical activity
- Patient education
- PT/OT
- Psychotherapy
- Nutrition

**Management is based on type of JIA*

COMPLICATIONS

- Osteoarthritis
- Osteoporosis
- Macrophage activation syndrome
- Growth issues
- Joint erosion
- Functional
- Psychological

Referral to ophthalmology for regular screening. Untreated **uveitis** can result in blindness!

Published January 2022
Alicia Synette (Medical Student, Memorial University), Dr. Todd Lambert (Pediatrician, Memorial University)
for www.pedscases.com

KAWASAKI DISEASE

Kawasaki disease is an **acute inflammatory vasculitis** of medium sized arteries.

CLINICAL FEATURES OF TYPICAL KAWASAKI DISEASE

Diagnostic criteria: **fever for ≥ 5 days** with ≥ 4 out of 5 of the following features:

Conjunctivitis	**Rash**	**Extremities**	**Adenopathy**	**Mucosal changes**
(bilateral, bulbar, non-purulent)	(any rash except vesicular)	Acute: palmar/plantar erythema and/or edema Subacute: periungual desquamation	(cervical, ≥ 1.5 cm, unilateral)	(strawberry tongue, cracked and erythematous lips, etc.)

Incomplete (atypical) Kawasaki disease: fever for ≥ 5 days with ≥ 2 of the above criteria. Assess for supplemental laboratory criteria.

Memory aid: *"WARM CREAM"*

OTHER POTENTIAL CLINICAL FINDINGS

Cardiovascular: coronary artery aneurysms, ↓ left ventricular function, pericarditis, myocarditis, mitral regurgitation, shock

Gastrointestinal: abdominal pain, diarrhea, nausea, vomiting

Neurological: irritability, facial nerve palsy, hearing loss

MSK: arthritis, arthralgia (pleocytosis of synovial fluid)

KD PHASES

- Acute: 1-2 weeks
- Subacute: 3-4 weeks
- Convalescent: 6-8 weeks

INVESTIGATIONS

- **Basic labs**: CBC+diff, lytes, Cr, CRP, ESR, ALT, albumin, urinalysis
- **Transthoracic ECHO** (rule out coronary artery aneurysms) at diagnosis and follow-up as indicated
- **ECG** (ST changes, pericarditis, myocarditis, etc.)
- **CXR** (rule out pulmonary pathology, rule out cardiomegaly)
- **Blood culture**, if clinically indicated
- Other **bacterial** and **viral testing** as clinically indicated
- **Multisystem inflammatory syndrome in children (MIS-C)**: above tests + COVID-19 PCR and serology, CK, ferritin, LDH, fibrinogen, D-dimer, PTT, INR, troponin, NT-proBNP

LAB FINDINGS

- Anemia
- ↑ platelets
- ↑ WBC
- Hyponatremia
- ↑ CRP
- ↑ ESR
- ↑ liver enzymes
- ↓ albumin
- Sterile pyuria

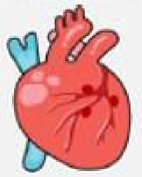
TREATMENT

- **Intravenous immunoglobulin (IVIG)**: 2g/kg x 1 dose, reassess after 24 hours, then consider a second dose.
  - ✓ If available, consider sending COVID-19 serology prior to IVIG administration.
- **ASA as per institutional guidelines** until normal follow-up ECHO.

COMPLICATIONS

Coronary artery aneurysms

- Untreated KD: 20-25%
- Treated KD: 1-3 %



Published March 2021
Katharine V. Jensen (Medical Student 2021, University of Alberta) and Dr. Hayley Turnbull (Pediatrician & Clinical Lecturer of Pediatrics, University of Alberta) for www.pedscases.com

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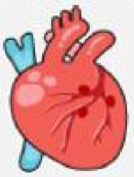

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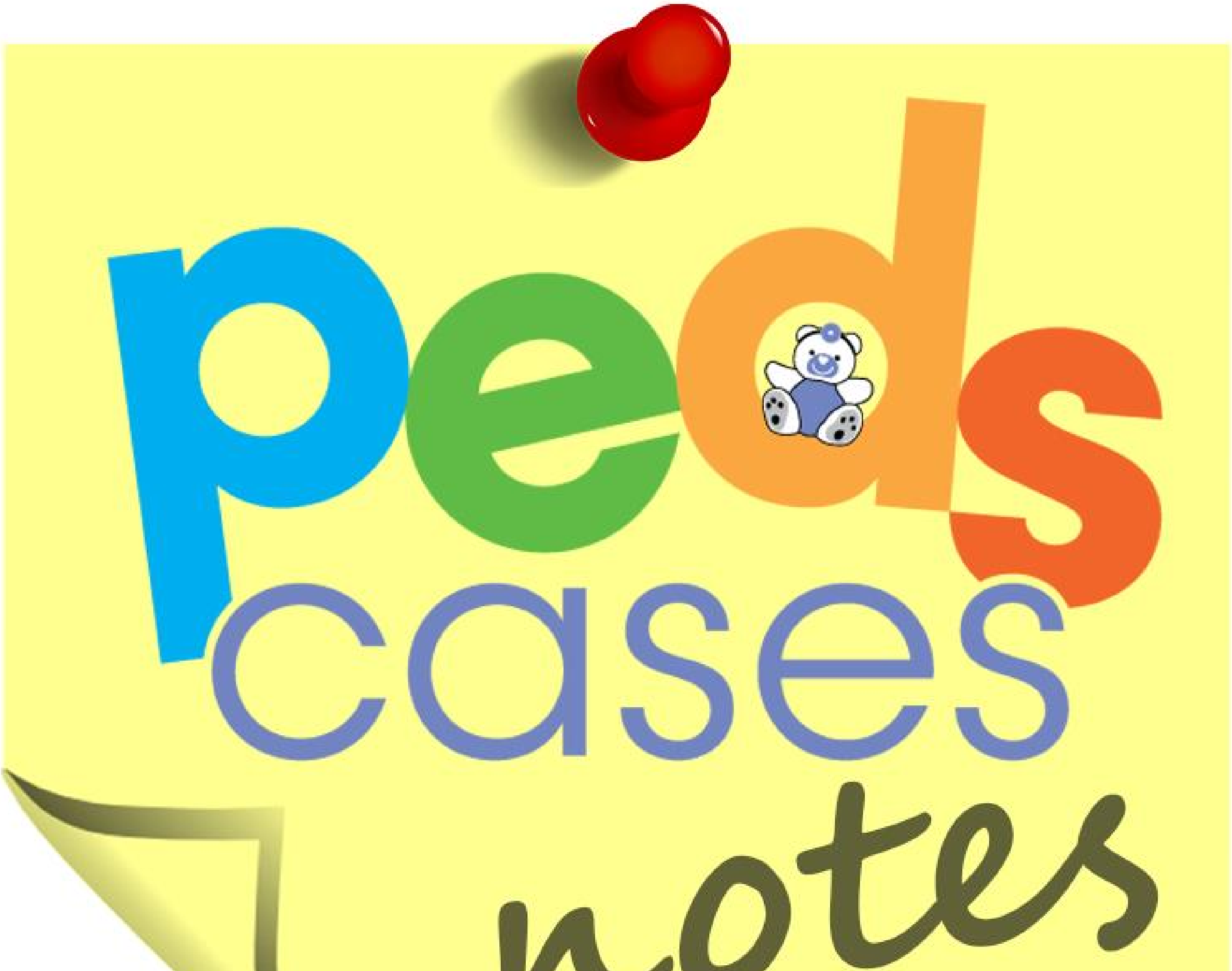

peds
cases
notes

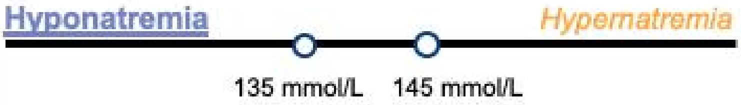
MAINTENANCE IV FLUIDS: AVOIDING HYPONATREMIA

Acute hyponatremia can lead to Cerebral Edema

Headache → Seizures

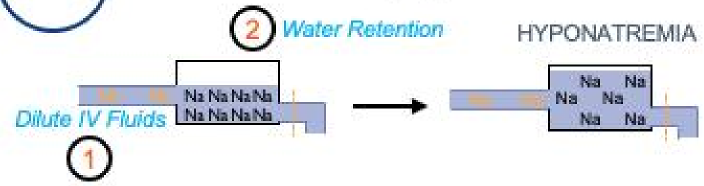
Lethargy → Cardiac Arrest

Acute: Less than 48 hours
Chronic: More than 48 hours



Hospitalized children who **receive maintenance IV fluids** are at an **increased risk of hyponatremia**

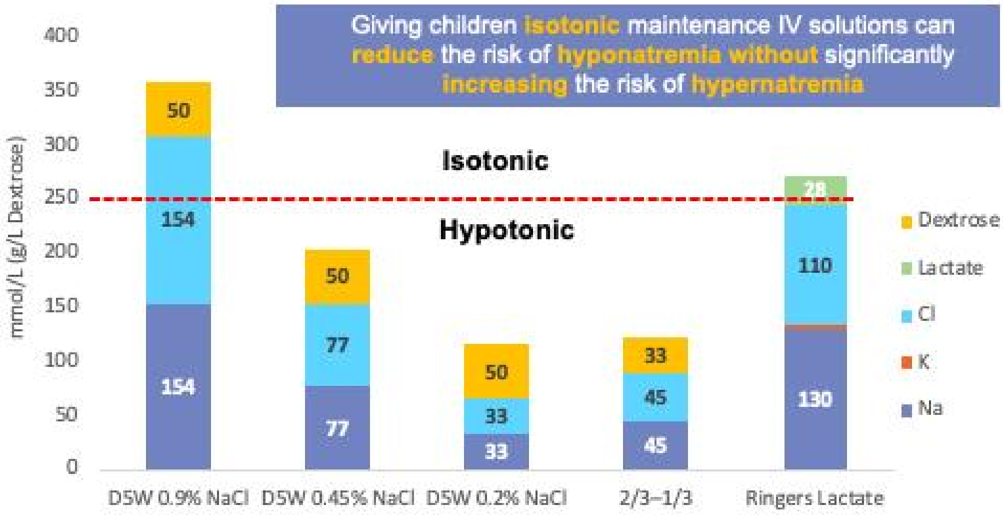
9-24% of these children *develop hyponatremia* to some degree. *Why?*



① It was routine practice to give children hypotonic IV solutions with 20-30 mmol/L of Na.

② Hospitalized children are at an increased risk of developing elevated ADH secretion due to nausea, stress, pain and medications like morphine

Giving children **isotonic** maintenance IV solutions can **reduce** the risk of **hyponatremia without** significantly **increasing** the risk of **hypernatremia**



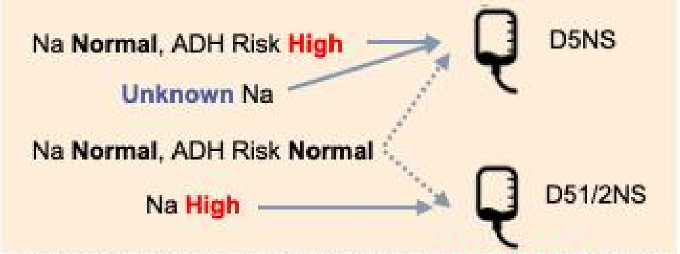
Monitor

- Baseline Electrolytes Glucose, Urea, Creatinine
- Regular Electrolytes
- Intake, Output, Weights
- Symptoms of hyponatremia

Recommendations

- All hospitalized children receiving IV fluids are at risk for hyponatremia
- Oral fluids are generally hypotonic and should be accounted for when assessing total fluid intake
- Infants and young children require dextrose with maintenance fluids (eg. D5NS) as they have limited glycogen stores.
- Clinicians should be equally cautious when prescribing IV fluids as they are when prescribing medications.

Prescription of IV Fluids for Maintenance Requirements in Hospitalized Children



Na **Normal**, ADH Risk **High** → D5NS

Unknown Na → D5NS

Na **Normal**, ADH Risk **Normal** → D51/2NS

Na **High** → D51/2NS

Hypotonic fluids less than 0.45% NaCl **should not** be used for fluid maintenance

Ringer's Lactate is generally **not appropriate** as a maintenance fluid in children

July 2019
Sparsh Shah (MD Student 2020, University of Toronto) for www.pedscases.com

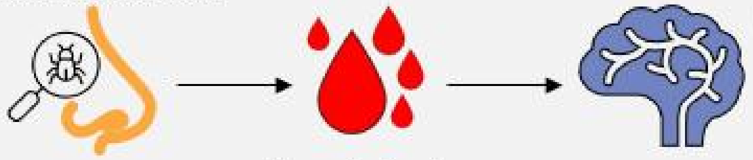
MENINGITIS

Meningitis is **inflammation of the meninges**.

RISK FACTORS

- Unimmunized
- Immunocompromised
- Age < 5 years old
- Crowded living conditions

PATHOGENESIS:



Bacteria colonize the nasopharynx → Bacteria invade into the bloodstream → Bacteria are transported to the CSF through the bloodstream

Bacteria replicate in the CSF

COMMON PATHOGENS

NEONATES (0-28 days)	*Group B strep* *E. coli* *Listeria*
1-3 MONTHS	*Group B strep* *S. pneumoniae* *N. meningitidis*
> 3 MONTHS	*S. pneumoniae (SP)* *N. meningitidis (NM)* *H. influenzae*

Viral: *enterovirus, parechoviruses, HSV*

CLINICAL PRESENTATION

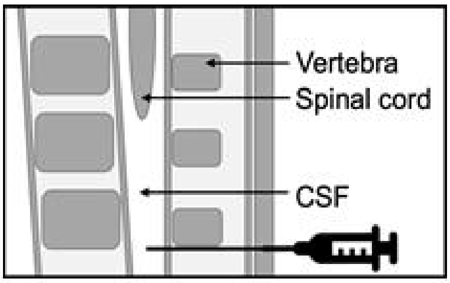
INFANTS	> 1 YEAR OLD	
Irritability Fever/hypothermia Seizures Lethargy Hypotonia Poor feeding	**Headache** **Fever** **Neck stiffness** Photophobia Nausea and vomiting	Confusion Irritability Anorexia ↓ LOC Seizures

INVESTIGATIONS

- **Lumbar puncture**
- Blood cultures
- CT or MRI head
- CBC, CRP, electrolytes, Cr, glucose

CSF ANALYSIS

	BACTERIAL	VIRAL
Glucose	↓	Normal
Protein	↑↑↑↑	↑
WBC	500-100,000	10-500
Predominant WBC	Neutrophils	Lymphocytes



__4 tubes__:
1. Gram stain & bacterial culture
2. Glucose & protein
3. Cell count & differential
4. Viral PCR

PREVENTION

- ✓ Haemophilus influenzae type b (Hib) vaccine
- ✓ Pneumococcal conjugate vaccine (PCV13)
- ✓ Pneumococcal polysaccharide vaccine
- ✓ Meningococcal conjugate vaccine

TREATMENT

__**Empiric antibiotic therapy**__:
- **Neonates**: ampicillin (*Listeria*) + cefotaxime (*GBS, E. coli*)
- **> 1 month**: ceftriaxone (*NM, SP*) + vancomycin (resistant *SP*) +/- ampicillin (immunocompromised)

- **Narrow antibiotic** based on susceptibilities
- **Acyclovir** if HSV suspected
- Consider adjuvant **corticosteroids**

COMPLICATIONS OF MENINGITIS

- Hearing loss
- Brain damage
- Seizures
- Gait problems
- Learning disabilities
- Memory difficulty
- Kidney failure

A **hearing test** is needed **within 1 month of discharge**.

Published November 2020
Katharine V. Jensen (Medical Student 2021, University of Alberta) &
Dr. Melanie Lewis (Pediatrician & Professor of Pediatrics, University of Alberta) for www.pedscases.com

STATUS EPILEPTICUS

Status epilepticus (SE) is a **medical emergency** that is associated with significant morbidity and mortality.

TYPES OF STATUS EPILEPTICUS

CONVULSIVE STATUS	NON-CONVULSIVE STATUS	FOCAL STATUS	ABSENCE STATUS
≥ 5 minutes of **continuous** convulsive seizure OR **≥ 2** discrete seizures **without return to baseline** OR repeated seizures for **≥ 30 minutes.**	**≥ 10 minutes** of continuous seizure OR **≥ 30 total minutes of ictal EEG activity** in any given hour. These patients are at risk for convulsive status.	Focal epileptic seizure that lasts **≥ 30 minutes** OR **repeated focal epileptic seizures** (≥ 30 minutes) with **incomplete recovery** between seizures.	Prolonged, generalized **absence** seizure that usually last for **hours** to **days**. Cardinal symptom is **altered level of consciousness**.

Convulsive status is the most common type of status epilepticus (SE). > 50% of SE episodes occur in children with **no prior seizure history**.

COMMON ETIOLOGIES OF STATUS EPILEPTICUS

ACUTE SYMPTOMATIC (17-52%)	REMOTE (16-39%)	OTHER
➢ CNS infection ➢ Metabolic (hypoglycemia, hyperglycemia, hyponatremia, hypocalcemia) ➢ Stroke ➢ Hemorrhage ➢ Non-compliance with AEDs ➢ Overdose ➢ Toxins	➢ Progressive neurodegenerative disorders ➢ Cerebral migrational disorders ➢ Perinatal hypoxic-ischemic encephalopathy ➢ Cerebral dysgenesis	➢ **Epilepsy** ➢ **Idiopathic (5-19%)** ➢ **Prolonged febrile convulsions (23-30%)** ➢ Trauma

HISTORY

- **Seizure history:** pre-ictal, ictal, and post-ictal phases. Ask about duration, focal symptoms, provoking events, and use of anti-convulsant medications.
- **Past medical history:** previous seizures or history of epilepsy or other neurological disorders.
- **Family history**: seizures or epilepsy.
- **Illness symptoms:** fever, nausea, vomiting, diarrhea, rash.
- **Trauma, accidental** and **non-accidental injury**
- Medications (**AED**)
- **Toxins**

PHYSICAL EXAM

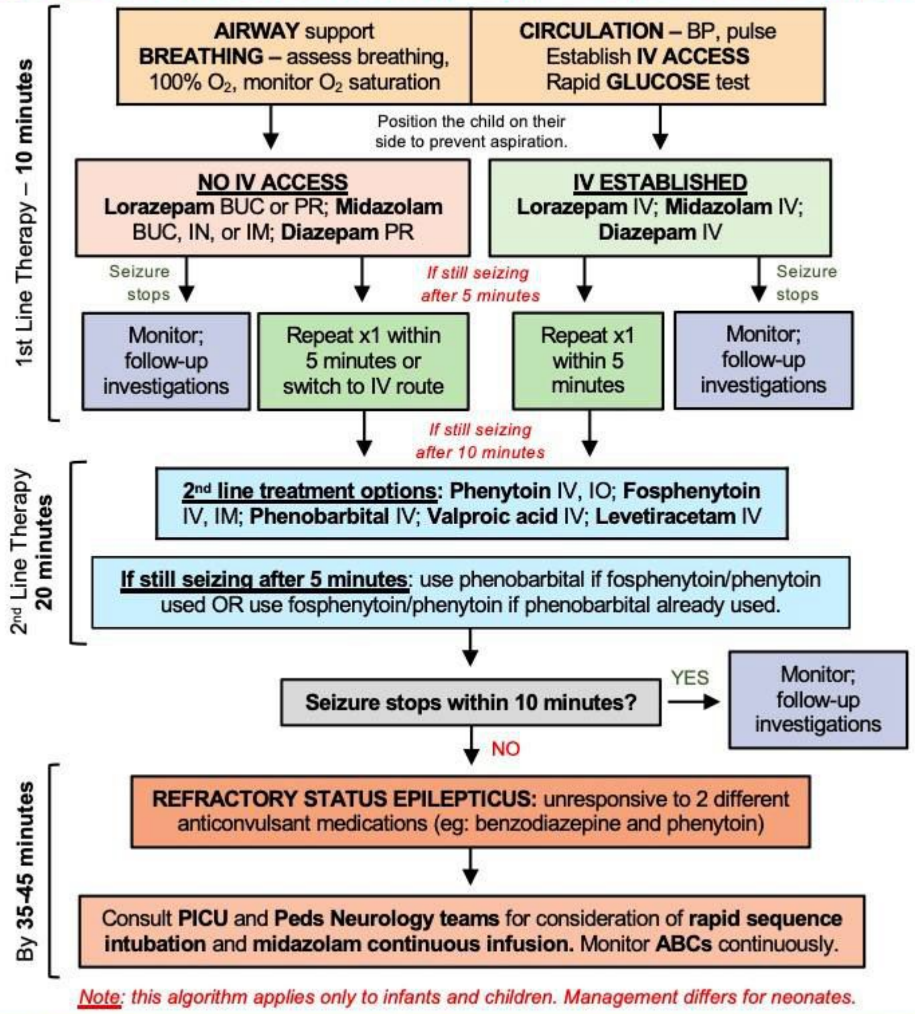
- **ABCs, vitals, level of consciousness, GCS**
- Rule out **CNS infections**
- **General physical exam:** source of infection (eg: otitis media, upper respiratory tract, lungs, GI tract, urinary tract, or sepsis).
- **Neurological exam**
- Indications of **toxidrome**

INVESTIGATIONS

- Glucose
- CBC with differential
- Electrolytes
- Ca^{2+}, Mg^{2+}, P
- Liver function tests
- Toxicology screen
- Anticonvulsant level
- Electroencephalogram (EEG)
- Head CT or MRI
- Urine, blood, CSF cultures

MANAGEMENT OF STATUS EPILEPTICUS IN HOSPITAL

GOAL: **stop the seizure** and **prevent brain injury**. Then determine the underlying cause.



1st Line Therapy – 10 minutes

AIRWAY support
BREATHING – assess breathing, 100% O_2, monitor O_2 saturation

CIRCULATION – BP, pulse
Establish **IV ACCESS**
Rapid **GLUCOSE** test

Position the child on their side to prevent aspiration.

NO IV ACCESS
Lorazepam BUC or PR; **Midazolam** BUC, IN, or IM; **Diazepam** PR

IV ESTABLISHED
Lorazepam IV; **Midazolam** IV; **Diazepam** IV

Seizure stops

If still seizing after 5 minutes

Seizure stops

Monitor; follow-up investigations

Repeat x1 within 5 minutes or switch to IV route

Repeat x1 within 5 minutes

Monitor; follow-up investigations

If still seizing after 10 minutes

2nd Line Therapy 20 minutes

2nd line treatment options: **Phenytoin** IV, IO; **Fosphenytoin** IV, IM; **Phenobarbital** IV; **Valproic acid** IV; **Levetiracetam** IV

If still seizing after 5 minutes: use phenobarbital if fosphenytoin/phenytoin used OR use fosphenytoin/phenytoin if phenobarbital already used.

Seizure stops within 10 minutes?

YES → Monitor; follow-up investigations

NO

By 35-45 minutes

REFRACTORY STATUS EPILEPTICUS: unresponsive to 2 different anticonvulsant medications (eg: benzodiazepine and phenytoin)

Consult **PICU** and **Peds Neurology teams** for consideration of **rapid sequence intubation** and **midazolam continuous infusion.** Monitor **ABCs** continuously.

Note: this algorithm applies only to infants and children. Management differs for neonates.

May 2020
Katharine V. Jensen (Medical Student 2021, University of Alberta) &
Dr. Natarie Liu (Neurologist & Epileptologist, University of Alberta) for www.pedscases.com

MANAGING POST-STROKE HEADACHES

The International Classification of Headache Disorders (ICHD-3) defines:

Acute post-stroke headache
"Headache developing in close temporal relation to other symptoms and/or clinical signs of stroke" and resolving within 3 months

Persistent post-stroke headache
Headache persistent for >3 months after stabilization of the stroke

Acute-Phase (< 3 months)

Management:
1st line: Acetaminophen

Other: Metoclopramide or Ondansetron

CAUTION: NSAIDs can inhibit platelet function and may increase systemic bleed risk when used acutely post-stroke.

Persistent-Phase (> 3 months)

Management:
Lifestyle Management:

SEEDS for:
1) Sleep Hygiene
2) Exercise 30 to 60 minutes, 3-5 times a week
3) Eat regular healthy meals
4) keep a headache Diary
5) Stress management (CBT, mindfulness, relaxation)

Nutraceuticals:
- Riboflavin, Coenzyme Q10, Magnesium, Melatonin

Interventional:
- Nerve blocks, botox injections, and neurostimulation

DIAGNOSIS

The first step in managing post-stroke headache is to rule out **secondary causes of headache**:

RED FLAGS
- **Systemic Symptoms**
- **Neurological Deficits**
- **Onset, sudden**
- **Onset age <5**
- **Progressive worsening**
- **Precipitated by Valsalva**
- **Postural Aggravation**
- **Papilledema**

IMAGING: A low threshold for repeat imaging should be applied. **CTA** (CT Angiography) and/or **MRI** are commonly used.

Abortive Therapies

1st line: Ibuprofen* and Acetaminophen

2nd line: Diclofenac* Powder

*NSAIDs exert antiplatelet effects - if the patient is on anticoagulants consider increased bleed risk

What not to use:
- *Triptans and Ergots* are vasoactive and should be avoided
- *Opioids* are associated with adverse effects including dependance and overdose

Daily Prophylaxis Therapies

Indication:
1. When a patient has one headache per week or more
2. Or when headaches are less frequent but very disabling

Management:
Topiramate, Amitriptyline, Propranolol

July 2024
Julia Craig (Medical Student, University of Alberta), Dr. Thilinie Rajapakse (Pediatric Neurologist, University of Alberta) for www.pedscases.com

MEASLES

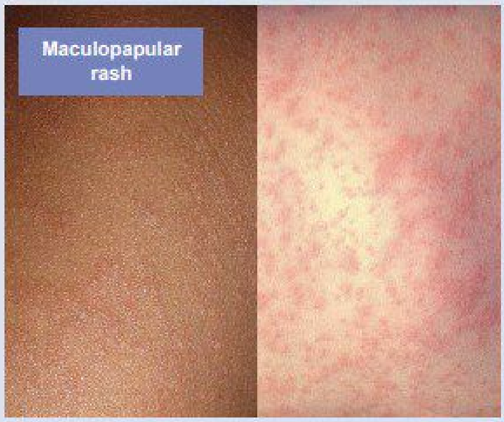
BACKGROUND

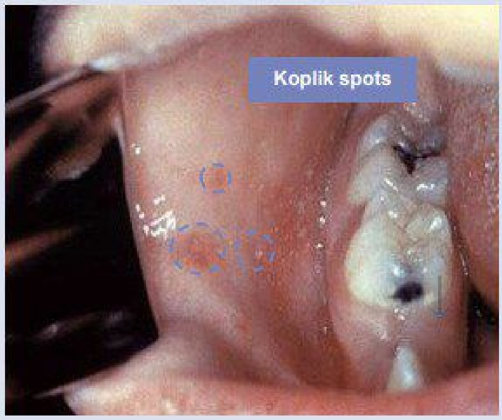
- ☐ Measles is one of the most contagious **airborne** diseases
- ☐ Caused by a **Paramyxovirus** (RNA) which mainly targets respiratory epithelial cells
- ☐ Risk factors for severe disease
  - Young infants (< 1 yo)
  - Unvaccinated
  - Nutrient deficient (Vitamin A)
  - Pregnancy
  - Immunosuppressed

MEDICAL URGENCY
One infected person can infect 9/10 close unimmunized contacts!

PROGRESSION

1. **Exposure**
2. **Incubation:** 6-21 days
3. **Prodromal stage:** 2-4 days
   - Fever
   - Cough
   - Coryza
   - Conjunctivitis – non-purulent
   - Koplik spots
4. **Exanthem:** cephalocaudal spread for 2-4 days (persisting fever may suggest a complication)
5. **Recovery:** cough can last for 1-2 weeks





PREVENTION

Outbreaks related to decreased herd immunity due to suboptimal vaccine coverage

MMR live attenuated vaccine:
1st dose at 12-15 months
2nd dose at 18 months or 4-6 years
(varying regimens worldwide)

Immunity in over 98% of individuals

COMPLICATIONS

- **Encephalitis (SSPE, ADEM)**
- **Pneumonia**
- **Otitis media**
- **Diarrhea**
- Myocarditis/Pericarditis
- Blindness
- Hepatitis
- Immune suppression

DIAGNOSIS

- ☐ Clinical diagnosis
- ☐ PCR viral RNA – throat and urine
- ☐ Serological testing (IgM, IgG antibody)

Reportable to public health

MANAGEMENT

No antiviral therapy, **supportive management** (hydration) with:

- ☐ **Isolation:** if hospitalized, negative pressure room + N95
- ☐ **Vitamin A:** in severe cases / malnourished

Postexposure prophylaxis:

- ☐ Vaccination within 3 days
- ☐ Immunoglobulin (Ig) in high-risk patients within 6 days

August 2024
Samir Gouin (Medical Student, McGill University), Dr. Marina Salvadori (Department of Pediatrics, McGill University), Dr. Mylene Dandavino (Department of Pediatrics, McGill University) for www.pedscases.com

MENINGITIS

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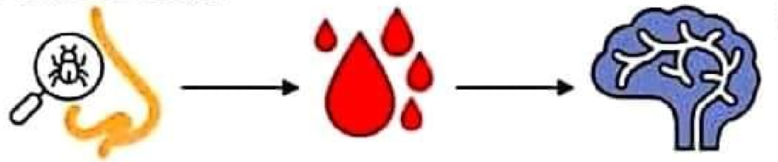
RISK FACTORS

- Unimmunized
- Immunocompromised
- Age < 5 years old
- Crowded living conditions

PATHOGENESIS:



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Viral: *enterovirus, parechoviruses, HSV*

CLINICAL PRESENTATION

INFANTS	> 1 YEAR OLD	
Irritability	**Headache**	Confusion
Fever/hypothermia	**Fever**	Irritability
Seizures	**Neck stiffness**	Anorexia
Lethargy	Photophobia	↓ LOC
Hypotonia	Nausea and vomiting	Seizures
Poor feeding		

INVESTIGATIONS

Lumbar puncture Blood cultures CT or MRI head	CBC, CRP, electrolytes, Cr, glucose

CSF ANALYSIS

	BACTERIAL	VIRAL
Glucose	↓	Normal
Protein	↑↑↑↑	↑
WBC	500-100,000	10-500
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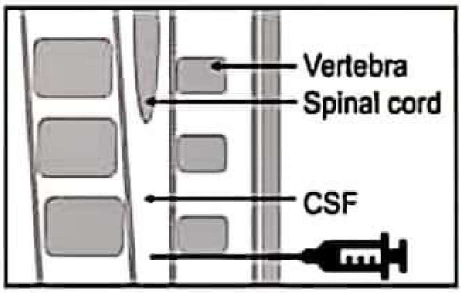

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3. Cell count & differential
4. Viral PCR

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- ✓ Pneumococcal conjugate vaccine (PCV13)
- ✓ Pneumococcal polysaccharide vaccine
- ✓ Meningococcal conjugate vaccine

TREATMENT

Empiric antibiotic therapy:
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- **> 1 month**: ceftriaxone (*NM, SP*) + vancomycin (resistant *SP*) +/- ampicillin (immunocompromised)

- **Narrow antibiotic** based on susceptibilities
- **Acyclovir** if HSV suspected
- Consider adjuvant **corticosteroids**

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- Brain damage
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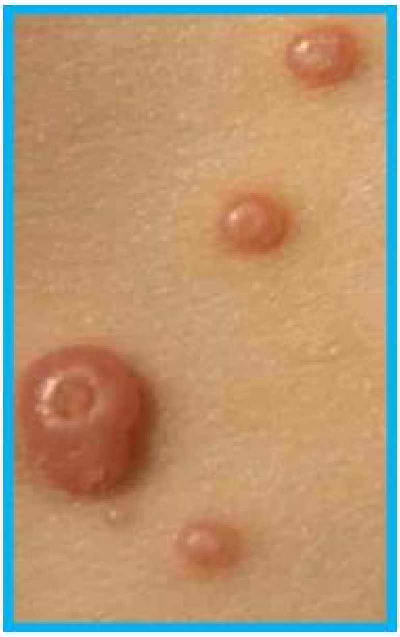
- Gait problems
- Learning disabilities
- Memory difficulty
- Kidney failure

A **hearing test** is needed **within 1 month of discharge**.

Published November 2020
Katharine V. Jensen (Medical Student 2021, University of Alberta) &
Dr. Melanie Lewis (Pediatrician & Professor of Pediatrics, University of Alberta) for www.pedscases.com

MOLLUSCUM CONTAGIOSUM



PATHOGENESIS

- Caused by **DNA poxvirus**.
- Spreads through **fomites, autoinoculation, and skin-to-skin.**
- **Swimmers** are regularly exposed to public pools with the virus.

CLINICAL PRESENTATION

- Primarily occurs in young **school-aged children**.
- It presents with multiple **small, skin-colored or pink, dome-shaped, umbilicated papules** (75% are umbilicated).

MANAGEMENT

Watchful waiting is appropriate for the majority of patients as on average most lesions resolve by 13 months.

- First-line therapies include cantharidin (most common), cryotherapy, curettage.

Physical destruction	**Curettage** **Cryosurgery** **Electrosurgery**
Topical therapy	Alkali agent (eg: potassium hydroxide) Blistering agent (eg: cantharidin) Imiquidmod (promotes immune response) Retinoic acid Salicylic acid

PREVENTION

- ✓ Minimize **shared items** (eg: towels, toys).
- ✓ To prevent spread, **wash hands, cover bumps, and avoid touching bumps.**

Published February 2021
Aryan Riahi (MD Student 2021, University of British Columbia) and Dr. J. Bergman, Paediatric Dermatologist
for pedscases.com

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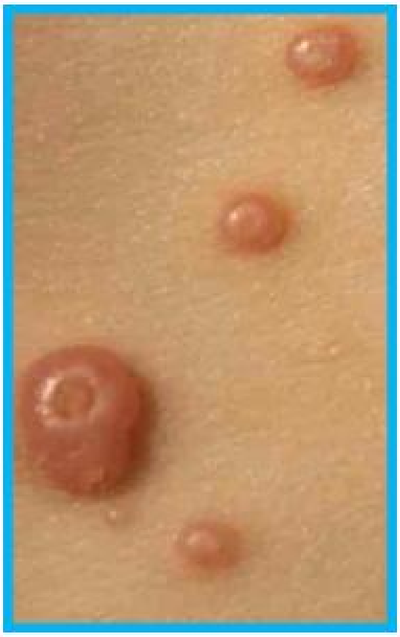

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EXAMPLES OF INNOCENT AND PATHOLOGIC MURMURS

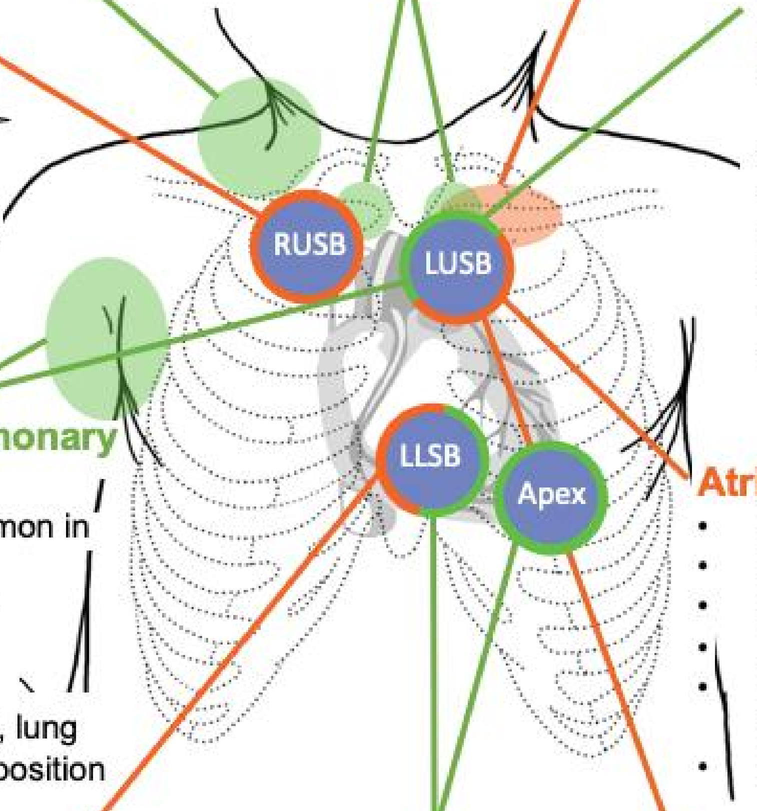
Carotid Bruit
- 2+ y/o, <>
- Short, mid systolic
- Over carotid arteries
- No change w/position

Cervical Venous Hum
- 2-7 y/o, R > L side
- Continuous rumbling at sternoclavicular junction
- ↑ w/turning head away from murmur + lift chin
- ↓ w/pressing over jugular vein or supine

Patent Ductus Arteriosus
- Any age
- Continuous, “machinery-like”
- Underneath L clavicle
- No change w/position

Aortic Valve Stenosis
- Any age
- Harsh, <>
- Ejection click
- May radiate to carotid vessels
- No change w/respiration

Pulmonary Flow murmur
- Older children/ adolescents
- Blowing,
- Early-mid systole
- Low-med pitched
- I, II, or III; <>
- Radiates to lung
- ↑ w/supine and inspiration



Peripheral Pulmonary Stenosis
- 0-6 mths, common in premature
- Blowing, short
- Mid systolic
- High pitched
- In LUSB, axilla, lung
- No change w/ position

Atrial Septal Defect
- Any age
- Radiates to lung
- Med-low pitched
- I, II, or III; <>
- Wide fixed splitting of 2nd heart sound
- No change w/position

Ventricular Septal Defect
- Any age
- Harsh
- Pansystolic or early-mid systole
- Low-high pitched
- II, III, or IV
- Other cardiac Sx

Still’s murmur
- 2-7 y/o
- Vibratory/musical
- Early-mid systole
- Med-low pitched
- I, II, or III
- Louder supine>sitting
- ↓ w/Valsalva

Pulmonary valve stenosis
- Any age, <>
- Radiates to lung
- Variable early systolic ejection click w/expiration only

November 2019
Sarah Park (MD student 2022, University of Alberta) for www.pedscases.com

NECROTIZING ENTEROCOLITIS

What is NEC?
Necrotizing Enterocolitis (NEC) is a potentially devastating gastrointestinal disease of neonates, characterized by bacterial translocation into the bowel wall, with associated inflammation and necrosis.

Incidence: 1-8% of NICU patients

RISK FACTORS

- Very low birth weight (<1500g)
- Prematurity
- Chorioamnionitis
- Mechanical ventilation
- Genetic predisposition
- Congenital anomalies
- Perinatal asphyxia, or anything that could result in hypoxia to the gut
- Formula feeding

NEC is associated with high **mortality (20-30%)**, and adverse long-term outcomes such as short bowel syndrome & poor neurodevelopmental outcomes.

INVESTIGATIONS

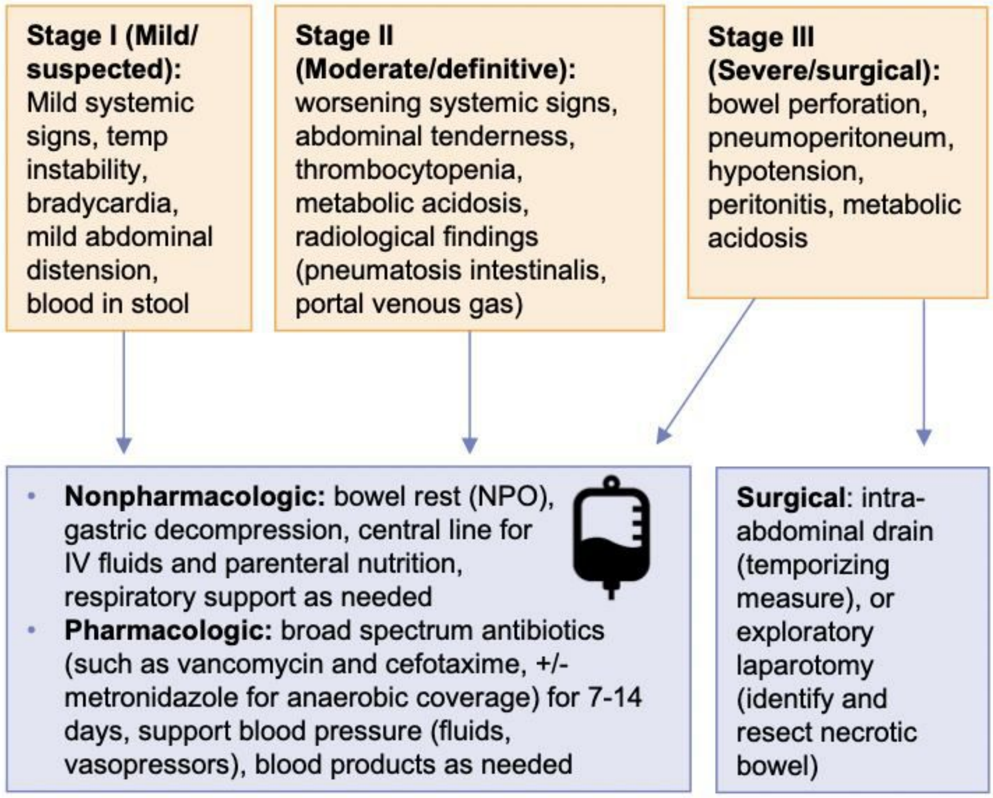
CBCdiff
Electrolytes
CRP
Blood gas
Blood cultures
Abdominal x-ray

PRESENTATION & DIAGNOSIS

- There is no test that is sensitive or specific for NEC
- Diagnosis is made in the context of clinical suspicion (feed intolerance, temperature instability, apnea, bloody stools, abdominal distention), lab abnormalities, and radiographic findings (see staging and management below)

STAGING & MANAGEMENT

Bell's modified staging criteria:



Stage I (Mild/ suspected): Mild systemic signs, temp instability, bradycardia, mild abdominal distension, blood in stool

Stage II (Moderate/definitive): worsening systemic signs, abdominal tenderness, thrombocytopenia, metabolic acidosis, radiological findings (pneumatosis intestinalis, portal venous gas)

Stage III (Severe/surgical): bowel perforation, pneumoperitoneum, hypotension, peritonitis, metabolic acidosis

- **Nonpharmacologic:** bowel rest (NPO), gastric decompression, central line for IV fluids and parenteral nutrition, respiratory support as needed
- **Pharmacologic:** broad spectrum antibiotics (such as vancomycin and cefotaxime, +/- metronidazole for anaerobic coverage) for 7-14 days, support blood pressure (fluids, vasopressors), blood products as needed

Surgical: intra-abdominal drain (temporizing measure), or exploratory laparotomy (identify and resect necrotic bowel)

PATHOGENESIS

- Multifactorial
- Intestinal immaturity
- Abnormal microbial colonization
- Altered vascular tone

PREVENTION

- Prevention is key!
- Early exposure to Mom's milk, or donor human milk if Mom's not available
- Careful progression to full volume feeds
- Probiotics
- Environmental modification (reduce light and sound, skin to skin care)

February 2023
Chelsea Gilbert (Medical Student 2022, University of Alberta), Dr. Bryan Dicken (Pediatric General Surgeon, University of Alberta) for www.pedscases.com

NEONATAL DISCHARGE COUNSELLING

KEY POINTS

Follow-up with your doctor in **24-72h**.

Seek medical attention **immediately** if your baby is unwell or has a **Temp >38ºC or <36ºC**.

Don't wait more than **4 hours** between baby's feeds.

Supplement with **Vitamin D** 400 units (1 drop) daily.

SAFE SLEEP (Strategies to Reduce Sudden Infant Death Syndrome)

Place your baby to sleep in a **crib or bassinet next to your bed**.

Breastfeeding can protect your baby.

Safe sleep environment: **firm surface, no pillows, toys, or bumper pads**.

Always place your baby on their **back to sleep**.

Babies should wear a onesie and or sleep sack. **Do not swaddle** in blankets.

Provide a **smoke-free** environment.

CHILD-PROOFING YOUR HOME

Lock away **medications** and cleaning products.

Remove **small objects** or broken parts that could be choking hazards.

Use **stair gates** and **window guards**. Baby walkers on wheels should never be used.

Cover **electrical outlets**; tuck away cords. **Secure furniture** to walls.

Keep infant away from **hot surfaces** and be careful when handling **hot liquids**.

BATH SAFETY

Never leave a baby **unattended** in a bathtub/ baby tub. Adjust hot water tank to **max of 49ºC** to prevent burns.

Bath seats, rings, and older children are not safe alternatives to **parental supervision**.

CAR SAFETY

Infants should be in a **rear facing** car seat in the **back**, ideally in the middle position, until at least **1 year and 10kg**.

Follow specific car seat instructions and ensure **proper installation**.

Ensure **latch is snug** over the chest at nipple line. Allow for two fingers between latch and chest.

Car seats are **not a safe place** for your newborn to sleep.

SAFETY OUTSIDE THE HOME

Babies who cannot sit without support cannot wear a personal flotation device. **1:1 adult:infant supervision is advised**.

Prevent **frostbite** by limiting time spent outdoors below **-15ºC**.

Published July 2021
Heather Halperin (Medical Student, University of Alberta) and Dr. L. Cynthia Gunaratnam (Pediatrician, University of Alberta) for www.pedscases.com

NEONATAL HYPOGLYCEMIA

Background

- Hypoglycemia is a common problem for neonates.
- Causes may include:
  - Lack of adequate **glycogen, amino acid,** and **lipid/fatty acid** stores.

Glucose Supply In Utero	Glucose Supply After Birth
• Placenta: Facilitated diffusion of glucose • Fetal insulin maintains homeostasis and euglycemia.	• No more constant supply from mother. • Initial drop within the first 2-3 hours after birth before subsequent increase.

Risk Factors

- **SGA/LGA/IUGR**
- **Diabetic mother**
- **Asphyxia**
- **Premature infant**

If present, check glucose levels at 2 hours of age

- *Other Risks:* Inborn errors of metabolism; Sepsis; Beckwith-Wiedemann Syndrome

DEFINITION OF HYPOGLYCEMIA:

Canadian Paediatric Society (CPS)	American Academy of Pediatrics (AAP)
< 72 HOURS OF AGE (TRANSITION): < 2.6 MMOL/L > 72 HOURS OF AGE (PERSISTENT): < 3.3 MMOL/L	< 4 HOURS OF AGE: < 40 MG/DL 4-24 HOURS OF AGE: < 45 MG/DL

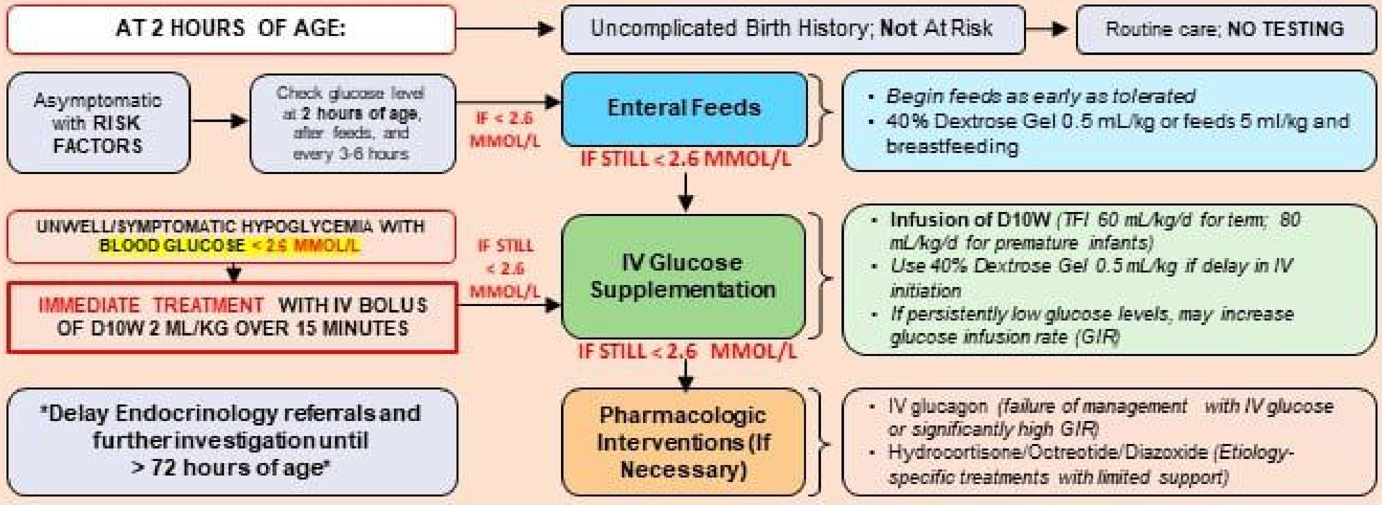
Signs & Symptoms

Neurogenic →	Neuroglycopenic
Sympathetic Nervous System Activation	Lack of CNS Glucose Availability
Diaphoretic	Apneic
Pale	Cyanotic
Irritable/Jittery	Hypotonic
Poor Feeding	Lethargic
Tachycardic	Coma
Unstable temperature control	Death

Prevention Techniques

- **Identification** of at-risk newborns
- **Frequent** and **early** breastfeeding
- Skin-to-skin contact
- Temperature **regulation**
- **Delaying** first bath
- Symptom **observation**
- **Prompt** treatment initiation

Screening & Management



AT 2 HOURS OF AGE:

Uncomplicated Birth History; **Not** At Risk → Routine care; **NO TESTING**

Asymptomatic with **RISK FACTORS** → Check glucose level at **2 hours of age**, after feeds, and every 3-6 hours → IF < 2.6 MMOL/L → **Enteral Feeds**

- *Begin feeds as early as tolerated*
- 40% Dextrose Gel 0.5 mL/kg or feeds 5 ml/kg and breastfeeding

IF STILL < 2.6 MMOL/L

UNWELL/SYMPTOMATIC HYPOGLYCEMIA WITH BLOOD GLUCOSE < 2.6 MMOL/L

IMMEDIATE TREATMENT WITH IV BOLUS OF D10W 2 ML/KG OVER 15 MINUTES

IF STILL < 2.6 MMOL/L → **IV Glucose Supplementation**

- **Infusion of D10W** *(TFI 60 mL/kg/d for tem; 80 mL/kg/d for premature infants)*
- *Use 40% Dextrose Gel 0.5 mL/kg if delay in IV initiation*
- *If persistently low glucose levels, may increase glucose infusion rate (GIR)*

IF STILL < 2.6 MMOL/L

Pharmacologic Interventions (If Necessary)

- IV glucagon *(failure of management with IV glucose or significantly high GIR)*
- Hydrocortisone/Octreotide/Diazoxide *(Etiology-specific treatments with limited support)*

Delay Endocrinology referrals and further investigation until > 72 hours of age

June 2020
Mekala Neelakantan (M4, Western Michigan University Homer Stryker M.D. School of Medicine) &
Dr. Janet Lioy (Children's Hospital of Philadelphia) for www.pedscases.com

NEONATAL HYPOTHERMIA

Hypothermia is defined as core body temperatures **below 36.5ºC**, however the consequences of hypothermia may occur at higher temperatures.
Neonates have a limited ability to thermoregulate, which increases the risk of hypothermia. Hypothermia in the neonatal population may have deleterious effects on **organ function.**

RISK FACTORS

Metabolic and endocrine dysfunction:
- Hypothyroidism
- Inborn errors of metabolism
- Hypoglycemia
- Addison's disease

Structural:
- Intracranial hemorrhage
- Gastroschisis
- Neural tube defects

Infection or sepsis

Birth in resource-poor environments:
- Higher rates of preterm and low-birthweight infants
- Inadequate delivery room environments
- Decreased thermoregulation during transport

Other:
- Premature birth
- Post-resuscitation
- Cold exposure

PATHOPHYSIOLOGY

There are 4 mechanisms through which a neonate loses heat:
- **Radiation**: infant is born into a setting containing cooler materials without direct contact, creating a gradient for temperature loss
- **Evaporation**: amniotic fluid evaporation
- **Conduction**: inadequately wrapped infant directly in contact with a cooler object
- **Convection**: surrounding cold air causes removal of heat from the infant

Clinical Presentation

- Cold temperature
- Acrocyanosis
- Irritability/lethargy
- Hypotonia
- Poor feeding
- Hypoglycemia

Categories of Hypothermia	Temp
Cold Stress	36-36.4ºC
Moderate Hypothermia	32-35.9ºC
Severe Hypothermia	< 32ºC

ADAPTATION TO COLD STRESS

Neonates adapt to the cold stress by:
- Increasing **heat production** by the heart, liver, and brain
- **Muscle flexion** to generate heat from voluntary movement and to minimize surface area
- **Peripheral vasoconstriction**
- Norephinephrine-directed **lipolysis of brown adipose** to create local heat reactions carried through the bloodstream to warm the body. This can be problematic as this causes a rapid increase in the overall metabolic rate, which can lead to hypoxia and hypoglycemia. **Premature infants may not have this capacity** as their bodies contain less brown adipose and overall fat stores.

It is crucial to recognize hypothermia to prevent the following consequences:

- Hypoglycemia
- Intraventricular hemorrhage
- Bradycardia
- Coagulation dysfunction
- Metabolic acidosis
- Hypoxia
- Apnea
- Sclerema neonatorum
- Mortality

PREVENTION

- **Warm delivery atmosphere**
- **Swaddling** and **drying** or **skin-to-skin** contact
- **Hats** to prevent heat loss from the scalp
- Continuous temperature regulation between **36.5-37.5ºC**
- Thermoregulation during **transfers** and **procedures**
- Special considerations for preterm infants:
    - **Increase temperature** of delivery room (25-26ºC)
    - **Polyethylene wrap** for pre-term infants <29 weeks
    - Preheat **radiant warmer**

THERAPEUTIC HYPOTHERMIA

Therapeutic hypothermia or total body cooling is the treatment of choice for neonates who meet criteria for hypoxic-ischemic encephalopathy. Hypothermia (33-35°C) is maintained for 72 hours until rewarming. This treatment is the only proven neuroprotective therapy for treatment of neonatal encephalopathy.

Published March, 2022
Morgan Gregg (Medical Student, University of Alberta), Dr. Nicole Anderson (Neonatal-Perinatal Fellow, University of Alberta) for www.pedscases.com

NEONATAL HYPOTONIA

Neonatal hypotonia is defined as **poor** tone in the muscles of the **trunk, limbs and/or face**. This means that the muscles provide little resistance when passively moved. Hypotonia can be categorized as central or peripheral (see clinical presentation). It can also be categorized as **axial or truncal**, predominantly affecting the neck and spinal muscles; **appendicular**, affecting predominantly the extremities; or **global**. It may be identified early in life when the newborn is unable to obtain a normal posture during movement or at rest.

CLINICAL PRESENTATION

CENTRAL HYPOTONIA *"Floppy but strong"*	PERIPHERAL HYPOTONIA *"Floppy but weak"*
☐ Hypotonic posture but may respond to external stimuli with appropriate power ☐ Reflexes normal or hyper-reflexive ☐ Often show other CNS abnormalities: decreased level of consciousness, seizures, apnea, feeding difficulties, and head shape abnormalities ☐ +/- Dysmorphic features	☐ Frog-leg posture ☐ Reflexes may be normal or hyporeflexive ☐ Diffusely low muscle bulk and/or congenital contractures ☐ Alertness and consciousness are preserved ☐ Symmetrical or asymmetrical pattern of weakness

HISTORY

- ☐ Timing and progression of hypotonia: acute vs chronic
- ☐ **Prenatal history:** abnormalities on US (polyhydramnios), chromosomal abnormalities, drug exposures, infections during pregnancy, gestational diabetes, hypertension, fetal movements.
- ☐ **Birth history:** gestational age, mode of delivery, forceps/vacuum, resuscitation, previous p9regnancies
- ☐ **Systemic illness:** GBS status, prolonged rupture of membranes, maternal fever, electrolyte abnormalities
- ☐ **Genetic causes:** family history, especially neurological

PHYSICAL EXAM

- ☐ **General Appearance:** Vital signs, level of consciousness, signs of systemic illness, contractures
- ☐ **Skin**: rashes, jaundice, cyanosis
- ☐ **HEENT**: dysmorphic features
- ☐ **Neuro**: fasciculations, CN exam, primitive and distal reflexes, observe spontaneous movements
- ☐ Horizontal and vertical suspension, traction response

DIFFERENTIAL DIAGNOSIS

Acute	Chronic

Systemic illness
☐ Sepsis/infection (ie. meningitis)
 	
Metabolic conditions
☐ Hypokalemia
☐ Hypophosphatemia
☐ Hypocalcemia
☐ Hypo/hypernatremia
 	
Genetic conditions
☐ Prader-Willi syndrome
☐ Down syndrome
	Genetic conditions
	☐ Prader-Willi syndrome
	☐ Down syndrome
	
	Neurological disorders
	☐ **Central**
	  ☐ Hypoxic-ischemic encephalopathy
	  ☐ Malformations of brain development
	  ☐ Intracranial bleeds or strokes
	☐ **Peripheral**
	  ☐ Spinal muscular atrophy
	  ☐ Myasthenia gravis
	  ☐ Congenital myopathies or muscular dystrophies

PRIMITIVE REFLEXES*

- ☐ Palmar grasp (a)
- ☐ Plantar grasp (b)
- ☐ Rooting (c)
- ☐ Moro (d)
- ☐ Asymmetric tonic neck (e)
- ☐ Galant (f)
- ☐ Landau (g)
- ☐ Parachute (h)
- ☐ Positive support (i)
- ☐ Placing and stepping (j)

*See note on Primitive Reflexes for more information.

INVESTIGATIONS

Guided by history and physical.
Systemic illness: septic workup, lytes, LFTs, ammonia, lactate
Neurologic causes: MRI brain +/- EEG; CK, electromyography/nerve condition study, muscle biopsy.
Genetic/metabolic: karyotype and microarray analysis, genetic testing for specific disorders, newborn metabolic screen and further metabolic testing as indicated.

(a)

(b)

(c)

(d)

(e)

(f)

(g)

(h)

(j)

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Morgan Gregg (Medical Student, University of Alberta) and Dr. Nicole Anderson (Neonatal-Perinatal Fellow, University of Alberta) for www.pedscases.com.

NEONATAL JAUNDICE

Jaundice: yellowing of the skin, scleral, and mucous membranes from a build up of bilirubin.

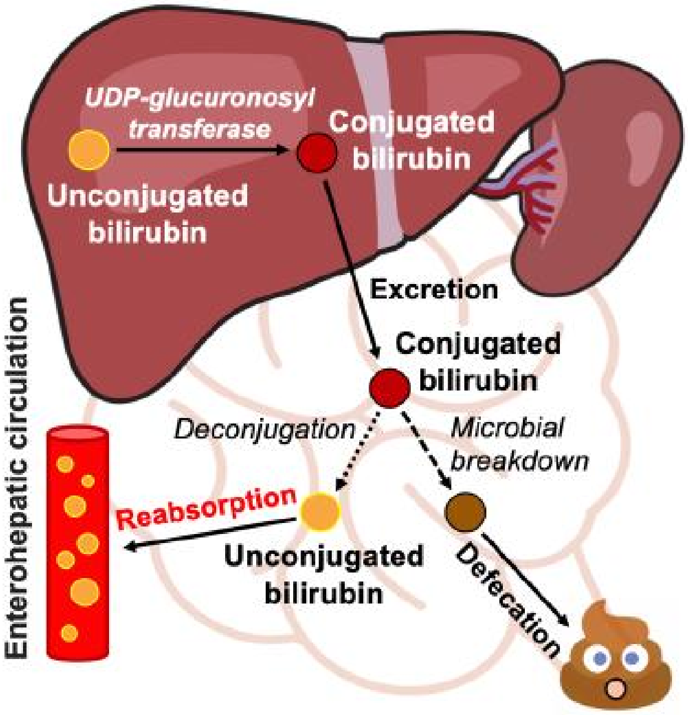
60% of **term infants** and **80%** of **preterm infants** develop jaundice in the 1st week of life, typically an **unconjugated hyperbilirubinemia**.

SCREENING

All infants in the ***first 24 hours*** of life:
- **Total serum bilirubin (TSB)**
- **Transcutaneous bilirubin**

RISK FACTORS

- Prematurity
- Cephalohematoma
- Bruising
- Dehydration
- Male sex
- Maternal age ≥ 25
- Asian or European descent
- Visible jaundice ≤ 24 hours
- Visible jaundice before discharge at any age
- Exclusive and partial breastfeeding
- Sibling with severe hyperbilirubinemia



UNCONJUGATED HYPERBILIRUBINEMIA – PHYSIOLOGIC JAUNDICE

BREASTFEEDING JAUNDICE	BREAST MILK JAUNDICE	PREMATURITY
- Early onset – **1st week** after birth - **Insufficient milk intake** leads to dehydration resulting in **hemoconcentration of bilirubin** - Fewer bowel movements **increases** the **enterohepatic circulation of bilirubin**	- Later onset – **after 1st week of life** - Bilirubin levels **peak during weeks 2-3 of life** - Can persist for **3-12 weeks** - Cause unknown - It is thought that substances in breast milk interfere with the breakdown of bilirubin	- Occurs in **preterm** infants (< 37 weeks) - More likely to require **phototherapy**

MANAGEMENT

- **Phototherapy** (use AAP normograms to determine the need for phototherapy – based on TSB and age in hours)
- **Continue breastfeeding**
- **Supplemental PO or IV fluids** (PO preferred over IV)
- Phototherapy makes bilirubin **water soluble** by inducing a **conformational change**
- Hyperbilirubinemia is treated to **prevent kernicterus/acute bilirubin encephalopathy**

PATHOLOGIC UNCONJUGATED HYPERBILIRUBINEMIA

HEMOLYTIC	NON-HEMOLYTIC
INTRINSIC - G6PD deficiency - Hereditary spherocytosis - Thalassemia **EXTRINSIC** - Drugs - Iso-immune (ABO, Rh) - Sepsis	- Sepsis - Hypothyroidism - Cephalohematoma - Gilbert - Crigler-Najjar **Work-up**: Coombs test, CBC with differential, blood smear, blood culture

CONJUGATED HYPERBILIRUBINEMIA

EXTRAHEPATIC	INTRAHEPATIC
- **Biliary atresia** - **Choledochal cysts** - **Perforated bile ducts** - **Tumour/mass** - **Cystic fibrosis** - **Galactosemia**	- **Infections**: hepatitis, TORCH, UTI, etc. - **Drugs**: eg. ceftriaxone, sulfonamides, etc. - **Genetic/metabolic**: eg. Alagille syndrome, etc.

⚠ **Must rule out biliary atresia!** ⚠
Initial investigation: **abdominal ultrasound**

Published March 2021
Katharine V. Jensen (Medical Student 2021, University of Alberta) and Dr. Jillian Popel (Pediatrician & Clinical Lecturer of Pediatrics, University of Alberta) for www.pedscases.com

pedscases notes

NEONATAL SEPSIS

Early onset neonatal sepsis (EONS): sepsis within the first 7 days of life.
Late onset neonatal sepsis (LONS): sepsis from 7-28 days of life.

HISTORY

- **Prenatal, pregnancy,** and **birth** history
- **Maternal serologies**
- **Risk factors for sepsis**
- **Neonatal complications** at delivery or prior to discharge home
- **Sick contacts**
- **Events leading up to current presentation**: sleep, waking, feeding, voiding, stooling

PHYSICAL EXAM

- **ABCs, vitals**
- **Airway**: patent?
- **Breathing**: ↑ WOB, apneic episodes, cyanosis
- **Circulation**: HR, BP, pulses, perfusion
- **Disability**: LOC, infant GCS
- **Exposure**
- **DEFG** = **don't ever forget glucose**

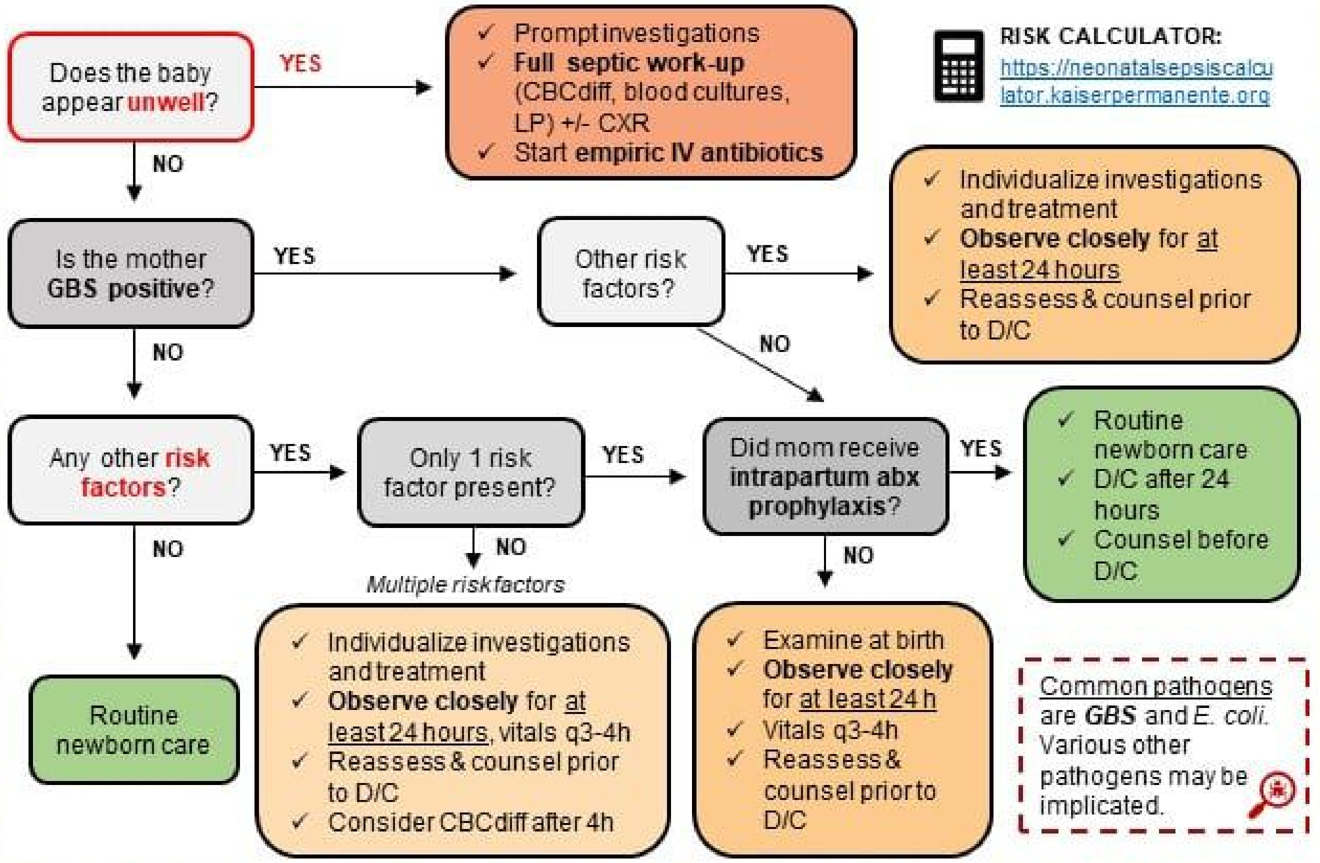
INVESTIGATIONS – FULL SEPTIC WORK-UP

- **Blood**: culture, CBC+diff, CRP
- **Urine** (catheter sample): culture, urinalysis
- **CSF**: culture, protein, glucose, cell count, viral PCR (HSV, VZV, and enterovirus).
- Consider: blood gas, CXR, respiratory virus panel, and swabs of any lesions.

RISK FACTORS FOR EONS

- **Prolonged rupture of membranes** (≥ 18 hours)
- **Maternal fever** (≥ 38°C)
- **Maternal GBS** colonization
- **GBS bacteriuria** anytime in pregnancy
- **Invasive GBS disease** in a previous infant
- **Chorioamnionitis**

MANAGEMENT OF TERM INFANTS (≥ 37 WEEKS) AT RISK FOR EARLY ONSET NEONATAL SEPSIS



Does the baby appear **unwell**?
YES →
- ✓ Prompt investigations
- ✓ **Full septic work-up** (CBCdiff, blood cultures, LP) +/- CXR
- ✓ Start **empiric IV antibiotics**

NO ↓

Is the mother **GBS positive**?
YES → Other risk factors?
YES →
- ✓ Individualize investigations and treatment
- ✓ **Observe closely** for at least 24 hours
- ✓ Reassess & counsel prior to D/C

NO → Did mom receive intrapartum abx prophylaxis?

NO ↓

Any other **risk factors**?
NO → Routine newborn care
YES → Only 1 risk factor present?
NO (Multiple risk factors) →
- ✓ Individualize investigations and treatment
- ✓ **Observe closely** for at least 24 hours, vitals q3-4h
- ✓ Reassess & counsel prior to D/C
- ✓ Consider CBCdiff after 4h

YES → Did mom receive **intrapartum abx prophylaxis**?
YES →
- ✓ Routine newborn care
- ✓ D/C after 24 hours
- ✓ Counsel before D/C

NO →
- ✓ Examine at birth
- ✓ **Observe closely** for at least 24 h
- ✓ Vitals q3-4h
- ✓ Reassess & counsel prior to D/C

RISK CALCULATOR: https://neonatalsepsiscalculator.kaiserpermanente.org

Common pathogens are ***GBS*** and *E. coli*. Various other pathogens may be implicated.

IV Antimicrobial Therapy: start **ampicillin** (*GBS* and *Listeria* coverage) + **gentamicin** (*E. coli* coverage) +/- **acyclovir** (if concerned about *HSV*). Ampicillin and gentamicin are generally preferred; however, *local antibiotic resistance patterns and special circumstances must be considered.*

October 2020
Katharine V. Jensen (Medical Student, University of Alberta) and Dr. Krystyna Ediger (Neonatal Perinatal Medicine Subspecialty Resident, University of Alberta) for www.pedscases.com

NEUROBLASTOMA

Epidemiology:

- Most **common malignancy in infancy** and most common pediatric extracranial solid tumor
- Median age at diagnosis is 18-19 months, with wide age range (e.g., in-utero diagnosis to patients in their 20s)

PRESENTATION

Depends on tumor's location and degree of differentiation (i.e., tumor behaviour ranges from spontaneous cell maturation to rapid metastasis).

Primary Tumor

- **Tumor can originate from any site within the SNS:**
  - Adrenal glands (>50%)
  - Abdominal paraspinal ganglia (24%)
  - Thoracic paraspinal ganglia (20%)
  - Neck (3%)
  - Brain (rarely)
- **Abdominal tumor:**
  - Palpable mass in the flank (hard, non-tender)
  - Distension
  - Abdominal pain
  - Constipation
- **Thoracic/cervical tumor**:
  - Dyspnea
  - Horner syndrome (ptosis, miosis, anhydrosis)
  - Stridor
  - Superior vena cava syndrome
- **Paravertebral tumor:**
  - Pain and numbness
  - Limb weakness
  - Sphincter dysfunction, loss of bowel/bladder function

Metastasis

- Metastasis is common at presentation
- May present with constitutional symptoms (e.g., anorexia, fever, weight loss)

Common sites of metastasis:

- **Bone/Bone Marrow:**
  - Bone pain
  - Limp
  - Pancytopenia
  - Periorbital ecchymoses secondary to orbital bone metastases
- **Liver**
- **Lymph nodes**
- **Skin**:
  - "Blueberry muffin" skin nodules (i.e., purple cutaneous nodules that blanch on palpation)

Paraneoplastic syndromes:

- Catecholamine production → hypertension, palpitations, and diaphoresis
- Vasoactive intestinal peptide production → secretory diarrhea (associated with hypokalemia)
- Opsoclonus myoclonus syndrome, presents as rapid, multidirectional eye movements, myoclonus of limbs or trunk, and/or ataxia

PATHOPHYSIOLOGY

Tumors arising from embryonic neural crest cells that normally form the sympathetic nervous system (SNS) and adrenal medulla

Sporadic (98%)	**Inherited/Familial** (2%)
MYCN amplification = most common somatic genetic mutation	ALK oncogene germline mutation = familial neuroblastoma

MYCN and ALK mutations are associated with aggressive tumor phenotype and poor prognosis

DIAGNOSIS

Laboratory

- ☐ Complete blood cell count with peripheral smear
- ☐ Complete metabolic panel, uric acid
- ☐ Coagulation panel
- ☐ Ferritin and lactate dehydrogenase levels
- ☐ Liver enzyme levels
- ☐ Urine vanillylmandelic acid and homovanillic acid

Radiology

- ☐ Initial: Chest radiograph or ultrasonography
- ☐ Urgent/emergency: CT scan of the chest, abdomen, and pelvis
- ☐ Preferred: MRI of the primary site and chest, abdomen and pelvis
- ☐ Metastatic: I-123 metaiodobenzylguanidine

Pathology

- ☐ Bilateral bone marrow aspirate and biopsy with immunohistochemistry analysis
- ☐ Tumor biopsy with immunohistochemistry analysis and DNA ploidy
- ☐ Segmental chromosomal alteration analysis
- ☐ Fluorescence in situ hybridization and polymerase chain reaction for MYCN and ALK

MANAGEMENT

Treatment, which may be observation only or include combo of surgery, radiotherapy, chemotherapy, autologous stem cell transplant, immunotherapy, is determined by numerous factors including stage, age and histology/genetics.

Staging can be determined by either of the following:

- Prior to treatment initiation, with radiology alone

OR

- After initial surgery, with use of histology

May 2023
Surabhi Sivaratnam (Medical Student, McMaster), Dr. Portwine (MD, McMaster) for www.pedscases.com

NORMAL PUBERTAL DEVELOPMENT

DEFINITION: PUBERTY

Developmental process leading to reproductive maturity & fertility. Characterized by:

- Secondary sexual characteristics
- Growth spurt
- Psychological, cognitive, social changes

ONSET OF PUBERTY

Girls: breast development

☐ Palpate to distinguish breast from adipose tissue

Boys: testicular enlargement

☐ Palpate to estimate size
☐ Testicular volume ≥ 3.0mL, or length ≥ 2.5cm

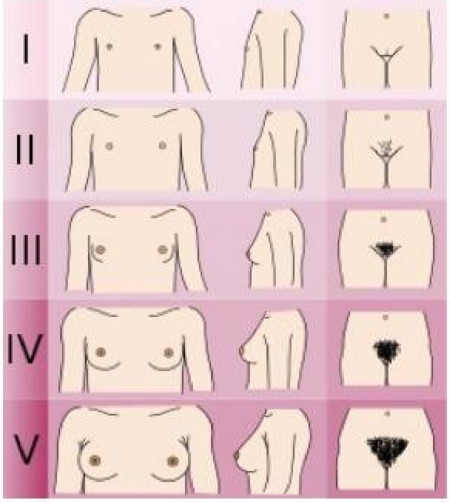
PRESENTATION

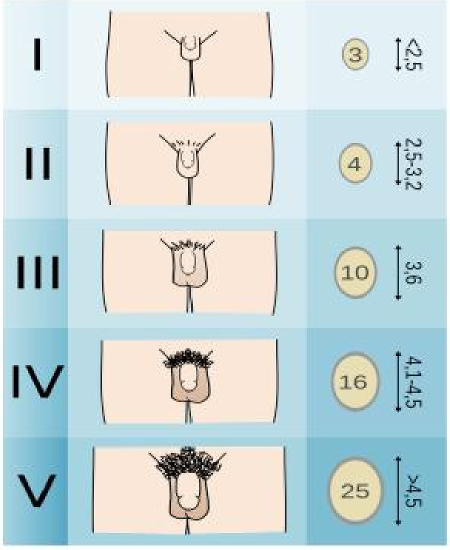
GIRLS	BOYS
Average age at onset: 10.5 yrs (range 8-13)	**Average age at onset: 11 yrs** (range 9-14)
Order 1. **Thelarche** ▪ Breast development 2. **Pubic hair** ▪ Body odour & mild acne often present 3. **Growth spurt** (2-3 yrs) ▪ Earlier in girls ▪ Between SMR stage 2-3 ▪ Peak height velocity: 6-10cm/yr 4. **Menarche** ▪ 2-2.5 yrs after pubertal onset ▪ Average age: 12-12.5 yrs ▪ May be irregular due to anovulatory cycles for first few years	**Order** 1. **Testicular enlargement** 2. **Pubic hair** ▪ Penile length increases, body odour, mild acne too 3. **Growth spurt** (2-3 yrs) ▪ Between SMR stage 3-4 ▪ Peak height velocity: 5-11cm/yr 4. **Voice change** ▪ Between SMR stage 4-5 **Other**: ~50% may experience pubertal gynecomastia, which resolves within 2 yrs

PHYSIOLOGY

Hypothalamus
↓ GnRH
Anterior Pituitary
↓ LH, FSH
Ovary/Testis
↓
↑ Production of sex steroids
Enlargement of gonads

SEXUAL MATURITY RATING (SMR) AKA. TANNER STAGING





September 2023
Selina Chow (Medical Student, University of Toronto), Dr Rhea D'Costa (Developmental Pediatrician, University of Toronto) for www.pedscases.com

NATIONAL RECOMMENDED IMMUNIZATION SCHEDULE

AGE	VACCINE	DISEASE PREVENTION
Birth	Hepatitis B	Hepatitis B virus
2 months	DTaP-IPV-Hib	Diphtheria, Tetanus, Pertussis, Polio, Haemophilus influenzae type b
	Pneumococcal conjugate (PCV13)	Pneumococcal disease
	Rotavirus (oral)	Rotavirus
4 months	DTaP-IPV-Hib	Diphtheria, Tetanus, Pertussis, Polio, Haemophilus influenzae type b
	Pneumococcal conjugate (PCV13)	Pneumococcal disease
	Rotavirus (oral)	Rotavirus
6 months	DTaP-IPV-Hib	Diphtheria, Tetanus, Pertussis, Polio, Haemophilus influenzae type b
	Hepatitis B	Hepatitis B virus
	Influenza (annually during flu season)*	Influenza*
	COVID-19 (2 doses, 8 weeks apart)**	COVID-19**
12 months	MMR	Measles, Mumps, Rubella
	Varicella	Chickenpox
	Pneumococcal conjugate (PCV13)	Pneumococcal disease
	Meningococcal C conjugate (Men-C-C)	Meningococcal disease
18 months	DTaP-IPV-Hib	Diphtheria, Tetanus, Pertussis, Polio, Haemophilus influenzae type b
4-6 years	DTaP-IPV	Diphtheria, Tetanus, Pertussis, Polio
	MMRV	Measles, Mumps, Rubella, Varicella
11-12 years	Tdap	Tetanus, Diphtheria, Pertussis
	HPV	Human Papillomavirus
	Meningococcal quadrivalent (Men-ACWY)	Meningococcal disease
14-16 years	Meningococcal quadrivalent booster (if needed)	Meningococcal disease

** Influenza vaccine recommended annually, starting at age 6 months*

*** COVID 19 - Recommended >6m. Specific vaccine used dependent on age of primary series. Please see the COVID-19 Canadian Immunization Guide for more specific and up to date information.*

***** Based on national recommendations. Schedules may vary depending on province. *****

November 2024
Lize-Mari Du Toit (Medical Student, University of Alberta), Dr. Karen Forbes (Pediatrician, University of Alberta) for www.pedscases.com

NORMAL VARIANTS OF SHORT STATURE

Definitions

- **Short stature**: height that is below the 3rd percentile
- **Familial short stature**: parents with short stature; normal growth velocity, proportionally normal weight, normal timing of pubertal onset, and normal bone age; predicted final height based on current growth pattern within expected range of mid-parental height
- **Constitutional delay of growth and puberty**: parents without short stature; normal pre-pubertal growth velocity, proportionally normal weight, late pubertal onset, delayed bone age, similar family history in one or both parents; should achieve normal final height without intervention

Measuring Stature

- 0 - 24 months = measure **length** (recumbent)
  - Calibrated length board, no shoes, eyes up, legs extended, toes upwards
- 2+ years = measure **height** (standing)
  - Wall-mounted stadiometer, no shoes, legs straight, looking straight ahead
- Normal growth in stature:
  - 0 - 24 months = rapid growth
  - pre-pubertal = slower consistent growth ~5 cm/year
  - puberty = rapid growth

History

- **Growth history** → onset of short stature, if child has always been short or recent growth velocity decrease, if weight is not proportional to height
- **Birth history** → gestation, birth weight and length, IUGR, neonatal hypoglycemia, jaundice, maternal illness, smoking
- **Developmental history** → developmental milestones, tooth eruption and loss, puberty onset
- **Nutrition** → calorie intake, feeding history
- **Social history** → indications of deprivation
- **Medications affecting growth** → stimulants, corticosteroids
- **Mid-parental height** using child's sex assigned at birth and biological parents' heights

$$\text{male} = \frac{\text{mother} + \text{father} + 13\text{ cm}}{2}$$

$$\text{female} = \frac{\text{mother} + \text{father} - 13\text{ cm}}{2}$$

- **Review of symptoms** → GI, cardiac, renal, thyroid diseases; neurological symptoms suspicious of tumours including headaches, vision changes

⚠ Decreased growth velocity is **more concerning** than a short absolute height.

⚠ Growth should be plotted on the **WHO Growth Charts for Canada**. For children with genetic syndromes, growth should be plotted on **syndrome-specific growth charts**.

Physical

- Full physical exam including cardiac, respiratory, GI, extraintestinal manifestations of IBD including eyes
- Plot height and growth velocity **every 6 months**
- Upper-to-lower segment ratio and arm span to evaluate for skeletal dysplasia
- Sexual maturity rating (Tanner stages)
- Features associated with genetic syndromes including Down syndrome, Turner syndrome, Noonan syndrome

Differential Diagnosis (With Examples)	Potential Investigations
Normal variants • Familial short stature • Constitutional delay of growth and puberty	• Bone age X-ray • No investigations is appropriate, given clinical context
Endocrine • Growth hormone deficiency • Hypothyroidism • Cushing's syndrome	• IGF-1, IGFBP-3, GH • T4, TSH • Cortisol, ACTH • LH, FSH, testosterone, estradiol • Glucose
Malabsorption • Celiac disease • Inflammatory bowel disease	• tTG-IgA • CRP, ESR, fecal calprotectin • Endoscopy, MR enterography
Chromosomal abnormalities • Skeletal dysplasia • Syndromes associated with short stature	• Genetic tests (molecular, chromosomal)

October 2023
Bryan Wong (Medical Student, Queen's University) and Dr. van Wylick (General Pediatrics, Queen's University) for www.pedscases.com

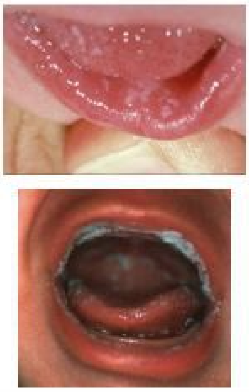
ORAL THRUSH

Oral thrush (oropharyngeal candidiasis) is a fungal infection of the oropharynx caused by an overgrowth of the yeast organism *Candida* species, most commonly *Candida albicans.*

Candida albicans is a normal part of the microbiome, but in certain populations, overgrowth may occur opportunistically.

PRESENTATION

- **White plaques on the buccal mucosa, palate, tongue, or oropharynx**
  - Infants < 6 months, most commonly
  - May be asymptomatic
  - Refusal to swallow or eat due to pain.
  - Older children may complain of a "cottony" feeling in their mouth or loss of taste.



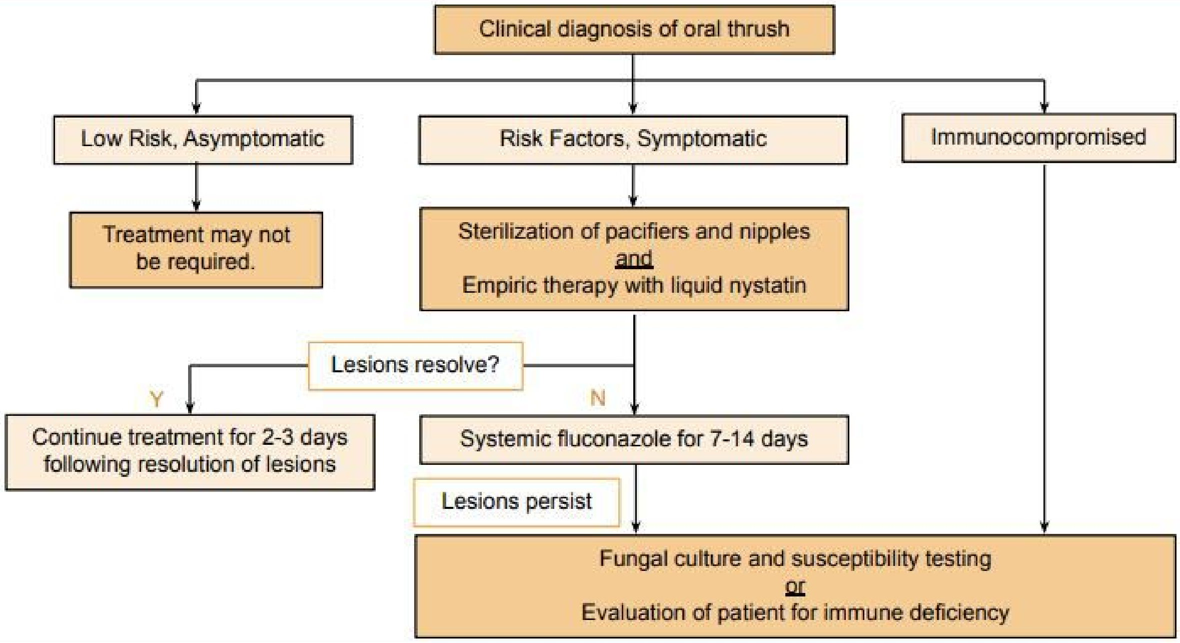
RISK FACTORS

- Immunodeficiency: primary vs. secondary (medications/chemotherapy)
- Recent antibiotic use
- Inhaled glucocorticoid use
- Radiation therapy
- Frequent/extensive pacifier use

DIAGNOSIS

- Clinical diagnosis in most cases
- Differential diagnosis:
- Milk tongue = white plagues limited to the tongue & wiping plaque with warm cloth will remove this
- In cases of treatment failure may rarely need to confirm diagnosis with Gram stain or potassium hydroxide (KOH) preparation.
- Diagnosis made based on presence of budding yeasts with or without hyphae.

MANAGEMENT



Clinical diagnosis of oral thrush

Low Risk, Asymptomatic → Treatment may not be required.

Risk Factors, Symptomatic → Sterilization of pacifiers and nipples and Empiric therapy with liquid nystatin → Lesions resolve?

Y → Continue treatment for 2-3 days following resolution of lesions

N → Systemic fluconazole for 7-14 days → Lesions persist → Fungal culture and susceptibility testing or Evaluation of patient for immune deficiency

Immunocompromised → Fungal culture and susceptibility testing or Evaluation of patient for immune deficiency

May 2024
Kylie Parent (Medical Student, University of Alberta), Dr. Jessica Foulds (Pediatrician, University of Alberta)
for www.pedscases.com

vnload

OSGOOD-SCHLATTER DISEASE

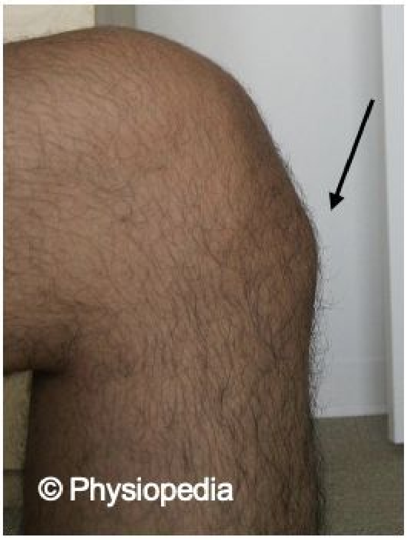
Osgood-Schlatter disease, alternatively referred to as osteochondrosis, tibial tubercle apophysitis, or traction apophysitis of the tibial tubercle, is a prevalent source of **anterior knee pain** among young **athletes**.

PRESENTATION

- **Atraumatic, insidious**, anterior knee pain
- Pain on palpation of **tibial tuberosity**
- Pain **worsens with physical activity**
- **Hypertrophy** at the tibial tuberosity
- **Tight quadriceps**

PHYSICAL EXAM

- **SEADS**: inspection for **s**welling, **e**rythema, **a**trophy, **d**eformity, and **s**cars
- Inspect for **bony protuberance** of the tibial tuberosity
- **Ely's test** for quadricep tightness



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PATHOPHYSIOLOGY

- **Overuse injury**
- Repeated **strain** and **microtrauma** lead to irritation
- Partial avulsion of the tibial tubercle apophysis may occur

RISK FACTORS

- **Male** gender
- Ages:
  - Boys 12-15
  - Girls 8-12
- Recent **growth spurt**
- **Repetitive activities** such as jumping and sprinting
- **Poor flexibility** of the quadriceps and hamstrings
- Common in children who play basketball, volleyball, running, gymnastic, or football

DIAGNOSIS

Osgood-Schlatter disease is a CLINICAL DIAGNOSIS

- Radiological imaging is generally unnecessary and is typically reserved for **severe cases** or situations where there is a **suspicion of avulsion**
- Consider **x-rays of bilateral knees** if symptoms are severe and/or persistent, there are concerns about a fracture or other structural issues, or the presentation is atypical

MANAGEMENT

- **Activity modification**
- **Cryotherapy**
- **RICE**: **r**est, **i**ce, **c**ompression, and **e**levation
- Nonsteroidal anti-inflammatory drugs (**NSAIDs**) PRN
- **Physiotherapy**
- **Stretching** the quadriceps, hamstrings, and vastus medialis
- Patellar **taping** or **bracing**

August 2024
Dr. Katharine V. Jensen (Pediatrics Resident, University of Alberta) and Dr. Jeanine McColl (Pediatric

OSTEOGENESIS IMPERFECTA (OI)

OI is a congenital disorder caused by genetic mutations resulting in collagen abnormalities. Patients often present with multiple fractures, hearing loss, and/or scoliosis.

Pathophysiology

- Mutation in COL1A1 or COL1A2 genes causing collagen cross-linking abnormalities
- Autosomal dominant (severe) and recessive (mild) forms
- Results in decreased production of type 1 collagen

PRESENTATION

Orthopedic findings

- Bone fragility
- Multiple fractures
- Poor bone remodeling
- Ligament laxity
- Short stature
- Scoliosis

Non-orthopedic findings

- Blue sclera
- Hearing loss
- Cardiovascular abnormalities
- Decreased pulmonary function

PHYSICAL EXAM

All types: Multiple fractures
Types II and III:

- Bowing of long bones
- Scoliosis
- Short stature

INVESTIGATIONS

Radiographs findings:

- Thin bone cortices
- Osteopenia
- Anterior bowing of the tibia
- Coxa vara
- Wormian bones

Consult Orthopedics, and consider Audiology, Cardiology, ENT, Genetics and Endocrinology as needed.

SILLENCE CLASSIFICATION OF OSTEOGENESIS IMPERFECTA

Type	Inheritance	Sclera	Features
Type I	Autosomal dominant Quantitative collagen disorder	Blue	Non-deforming type. The most common type of OI. Often presents at 3-5 yrs. Hearing deficits and brittle teeth are common. ≤1 fracture per year.
Type II	Autosomal dominant Qualitative collagen disorder	Blue	**Deforming type**, potentially lethal within the perinatal period. 3+ fractures per year.
Type III	Autosomal dominant Qualitative collagen disorder	White	**Deforming type**, often present with fractures at birth and significantly short stature. Hearing loss is common. 3+ fractures per year.
Type IV	Autosomal dominant Qualitative collagen disorder	White	Non-deforming type. Long bone bowing, vertebral fractures, and hearing loss are common. ≤1 fracture per year.
Type V-XXII	Non-collagen mutation	White	Non-deforming type. Present with multiple fractures. Hearing loss is uncommon. ≤1 fracture per year.

Types I-IV are the most common. Types V-XXII are rare and not defined separately within this resource.

MANAGEMENT

Goals of Treatment: Maximize mobility, pain control, fracture prevention and management

Fracture Prevention

- Bisphosphonates
- Growth Hormone
- Bracing
- Physiotherapy

Acute Fracture Management

Fractures can be managed non-operatively (splint/cast) or operatively

- Fractures management should follow the standard of practice in your jurisdiction.

Deformity Correction

Operative: Realignment osteotomy using rod fixation

- Indicated when severe deformity is present
- Telescoping or non-telescoping devices may be used

March 2024
Aspen Lillywhite (Medical Student, University of Alberta) and Dr. Elizabeth Pedersen (Pediatric Orthopedic Surgeon, Stollery Children's Hospital) for www.pedscases.com

OSTEOMYELITIS

Inflammation of the cellular and extracellular matrix of bone due to **infection with a microbial pathogen.**

Acute osteomyelitis: diagnosis of bone infection within 4 weeks after the onset of clinical manifestations.

Chronic osteomyelitis: protracted, indolent disease process with sequestrum and/or relapse of infection more than 4 weeks after the initial diagnosis and treatment.

DIAGNOSIS

Gold Standard: microbiological & pathological assessment of bone sample *(only if need for debridement)*

- ☐ MRI with gadolinium enhancement
- ☐ X-ray may not be helpful as findings lag 7-10 days after symptom onset

COMMON BACTERIA

In children older than 1 month:

1. *Staphylococcus aureus* (up to 33% are MRSA)
2. *Streptococcus pyogenes*
3. *Streptococcus pneumoniae*
4. *Kingella kingae**

**higher rate of infection in younger children*

CLINICAL PRESENTATION

- ☐ Pain involving a bone or joint
- ☐ Lack of weight bearing & decreased use of affected extremity
- ☐ Fever
- ☐ Focal swelling, tenderness, warmth, erythema when metaphyseal and periosteal infection with abscess

⚠ **Pain near the buttock or pelvic area?**

- ☐ High degree of suspicion for osteomyelitis in the pelvic bone or ileum!
- ☐ Physical findings are very difficult to elicit.

⚠ Septic arthritis or bone/soft tissue abscess? Needs debridement!

INITIAL WORK-UP

- ☐ CBC
- ☐ Blood Culture
- ☐ CRP
- ☐ +/- ESR
- ☐ Radiograph *(rule out trauma)*

PATHOPHYSIOLOGY

Commonly acquired through hematogenous (bacteremia), through direct inoculation (trauma or recent procedures), or via spread from adjacent soft tissue or synovial fluid infection.

With hematogenous osteomyelitis, organisms enter the bone through nutrient arteries and proliferate in venous sinusoids.

Children have a rich vascular supply in their bones especially around the growth plates.

Common sites: metaphysis in long tubular bones.

DIFFERENTIAL DIAGNOSIS

Transient synovitis of hip
Fracture/Trauma
Cellulitis
Hematologic malignancy
Juvenile idiopathic arthritis
Bone neoplastic lesion
Lyme disease arthritis
SLE

APPROACH TO MANAGEMENT

If fully immunized, empiric antibiotics

- ☐ First generation cephalosporin
- ☐ Common: Cefazolin 100-150mg/kg/day IV q6-8h

If risk of MRSA

- ☐ Same as above and ADD Vancomycin until further testing confirms MRSA negative

If age <4yo, and unimmunized

- ☐ Empiric therapy with IV cefuroxime to also add coverage for *H. influenzae*

With cultures and susceptibilities

- ☐ Switch to targeted therapy specific to pathogen

Post hospital stay

- ☐ Switch to PO antibiotics with clinical improvement and decreased CRP (usually within a week) or following consultation with Pediatric Infectious Diseases.
- ☐ Treat for a total of 3-4 weeks with antibiotics if uncomplicated.

CONSULTS

- ☐ ***In the community?*** - Referral to hospital for further assessment and initiation of IV antibiotics
- ☐ ***Abscess, septic joint, complicated course?*** - Consult Pediatric Orthopedic Surgery
- ☐ ***Atypical bacteria or fungi?*** - Consult Pediatric Infectious Disease

May 2023
Shaharyar Ahmad (Medical Student 2023, University of Alberta) & Dr. Sarah Forgie (Pediatric Infectious Diseases, University of Alberta) for www.pedscases.com

PATELLOFEMORAL PAIN SYNDROME

A broad term to describe **pain in the front of the knee and around the patella.** Also known as "runner's knee" or "jumper's knee".

RISK FACTORS

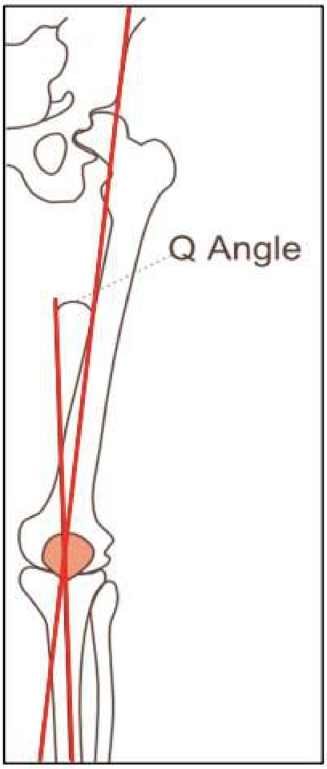
- Female
- Weak vastus medialis
- Weak core
- Tight hamstrings
- Tight iliotibial (IT) band
- Increased Q angle

PRESENTATION

- Anterior knee pain with **activity**
- Anterior knee pain after **sitting for prolonged periods** with bent knees
- Difficulty going up or down stairs
- Difficulty with squatting or lunges
- Pain related to a **change in activity intensity, playing surface,** or **equipment**

PHYSICAL EXAM

- **SEADS**: inspection for **s**welling, **e**rythema, **a**trophy, **d**eformity, and **s**cars
- **Alignment**
- Strength, flexibility, and tone
- Knee stability
- ROM of knees and hips
- **Patellar grind**
- **Clark's test** (press patella inferiorly while patient contracts quads, positive test is pain)



PATHOPHYSIOLOGY

PFPS occurs when nerves sense pain in the soft tissues (tendons, patellar fat pad, synovial tissues) and bone around the patella, caused by a muscle imbalance. **Vastus lateralis is often stronger than vastus medialis causing malalignment of the patella as it tracks along the trochlear groove of the femur.**

- **Overuse:** repeated stress on the knee from physical activity
- **Patellar malalignment:**
  - Malalignment of the leg between the hips and the ankles
  - Muscular imbalances or weaknesses, especially in the quadriceps muscles at the front of the thigh

DIAGNOSIS

Patellofemoral syndrome is a CLINICAL DIAGNOSIS

- Generally, investigations are unnecessary; however, in situations where a definitive diagnosis remains elusive, consider imaging
- **X-ray:** to rule out other potential causes of knee pain, such as arthritis or structural abnormalities in the knee joint
- **MRI**: if concerned about soft tissue injury, such as injury to ligaments, tendons, and/or muscles

MANAGEMENT

- **Activity modification** (e.g. avoid overstressing the knee, run on soft surfaces)
- **RICE: r**est, **i**ce, **c**ompression, and **e**levation
- Nonsteroidal anti-inflammatory drugs (**NSAIDs**), such as ibuprofen and naproxen, help reduce swelling and relieve pain
- Referral to **physiotherapy**
- If elevated BMI, **weight loss** may alleviate pain
- Quadricep & core **strengthening exercises**
- Hamstring and iliotibial band **stretching exercises**
- Appropriate **footwear**

August 2024
Dr. Katharine V. Jensen (Pediatrics Resident, University of Alberta) and Dr. Jeanine McColl (Pediatric Rheumatologist, University of Calgary) for www.pedscases.com

PEDIATRIC ADMISSION ORDERS

Note the ***date*** *and* ***time.*** *Ensure* ***correct patient identification*** *using multiple identifiers.*

ADMIT to General Pediatrics under the **attending physician**. Specify the admitting **team, location,** and **level of care**. Update medical history, mediations, and allergies. **A**

DIAGNOSIS: provide the **working diagnosis/reason for admission** to hospital. **D**

DIET: order an **age-appropriate diet** based on the child's presentation, considering available options such as breastfed, expressed breast milk (EBM), or formula for infants. For children, purees, regular diet, clear fluids, full fluids, easy-to-chew, low sodium, diabetic, etc. Specify the **route of administration** (NG tube, G-tube, PO) and consider any **dietary restrictions**. If the child is scheduled for upcoming procedures, sedated imaging, or surgery, consider the need for **NPO (nothing by mouth) status** and communicate this clearly in the dietary orders. **D**

ACTIVITY: indicate any necessary activity restrictions based on the child's medical needs. Include details such as **limitations on physical activities, play, or mobility**. Consider any precautions such as **seizure or reflux precautions**. **A**

VITAL SIGNS: specify the **frequency** for monitoring vital signs based on the child's condition and the level of acuity (eg. q1h, q4h, q8h, qShift). Include **parameters** for temperature, heart rate, respiratory rate, SpO2, and blood pressure. The decision to use **cardiorespiratory monitors** depends on the severity of the child's condition. **Continuous pulse oximetry** may be sufficient depending on the presentation. **V**

IV/NG FLUIDS: order fluid type, route, and rate.
INTAKE AND OUTPUT: closely monitor intake and output, specify frequency, and notifying the physician if **urine output is < 1 cc/kg/hr**.
INVESTIGATIONS: order appropriate investigations based on the patient's condition, including but not limited to blood tests, imaging studies, cardiac investigations (ECG, echocardiogram), microbiological tests (blood cultures, urine culture, RPP, throat swab, etc.), and others, specifying the **urgency** (timed vs. STAT).
ISOLATION: specify the appropriate isolation precautions based on the child's medical condition (**airborne, contact, droplet, contact & droplet, sporicidal,** etc.). **I**

DRUGS: ensure continuity of **home medications** as appropriate. Order any needed **PRN medications** and **essential treatments** based on the patient's presentation.
DRAINS: note any drains, monitor drain output, and drainage characteristics.
DRESSINGS: specify the type of dressing as well as the frequency of changes. **D**

Do not forget to ***sign your orders****.*

March 2024
Dr. Katharine V. Jensen (Pediatrics Resident, University of Alberta) and Dr. Karen Forbes (Professor of Pediatrics, University of Alberta) for www.pedscases.com

PEDIATRIC ANXIETY

ANXIETY DISORDERS IN PEDIATRICS

- Anxiety disorders are **common** in pediatrics
- Median age of onset: **11 years old**
- Recognizing anxiety in children may be **difficult**
- Often manifests as **somatic complaints** (e.g.: headaches, stomach aches, nausea, etc.)

PEDIATRIC ANXIETY ASSESSMENT

- **Screening tool**: Screen for Child Anxiety Related Disorders (SCARED)
- Interview appropriate to **age level**
- **Prenatal, birth,** and **developmental history**
- **Collateral** from primary caregivers
- Input from **community supports** such as school, therapists, government agencies (e.g.: CFS)
- **Psychoeducational** assessment
- **Trauma history**

GENERALIZED ANXIETY DISORDER (GAD)

- **3-5%** of children & adolescents
- Mean age of onset: **late adolescence**
- **F > M** (2:1)
- Left untreated, GAD may worsen and can create moderate to severe **impairment** in life functioning in adulthood
- Chronic, generally **lifelong** condition

DSM-5 Criteria:

- **Excessive worry**, more days than not, for **≥ 6 months**
- **Difficult to control** worry
- Associated with ≥ 1 of the following (vs. ≥ 3 for adults):
  1. Restlessness/ on edge
  2. Easily fatigued
  3. Difficulty concentrating
  4. Irritable
  5. Muscle tension
  6. Sleep disturbance
- Causes **impairment** (e.g.: school)

PANIC DISORDER

- **Underdiagnosed** in pediatrics
- **F > M** (2-3:1); Onset in **late adolescence**
- Attacks last on average **10 minutes** (rarely > 1 hour)
- Can be diagnosed with or without **agoraphobia**

DSM-5 Criteria:

- **Recurrent unexpected panic attacks** (sudden intense fear, peaking within minutes, and consisting of *≥ 4 of the following*: palpitations/tachycardia, diaphoresis, trembling, SOB, choking, chest discomfort, nausea, dizziness, hot/cold, paresthesias, derealization, fear of losing control, fear of dying)
- At **least 1 attack** followed by **≥ 1 month** of one or both of persistent **concern/worry about future attacks or their consequences**, AND/OR significant **maladaptive behaviours**

SEPARATION ANXIETY DISORDER

DSM-5 Criteria:

- **Intense fear** or **anxiety** on separation that is **developmentally inappropriate**
- **Distress** when experiencing or anticipating separation
- **Worry about losing** attachment figures
- Worry about an **untoward event** that would **lead to separation** (e.g.: kidnapping, accident, illness, death, etc.)
- **Reluctance or refusal to go out**
- **Afraid to be alone** or without the attachment figure
- **Refusal to sleep** without being near the attachment figure
- **Nightmares** regarding separation
- **Physical complaints** (e.g.: headaches, stomach aches, nausea, vomiting) when separated
- Lasting **≥ 4 weeks**
- Causes **distress** and **impairment**

OBSESSIVE COMPULSIVE DISORDER (OCD)

DSM-5 Criteria:

- Presence of obsessions, compulsions, or both:
  - **Obsessions**: recurrent and persistent *thoughts, urges, or images*, causing marked anxiety or distress
  - **Compulsions**: *repetitive behaviors* or *mental acts* that the individual feels driven to perform in response to an obsession. The behaviors or mental acts are aimed at preventing or reducing anxiety or distress
- Young children **may not be able to articulate the aims** of these behaviors or mental acts
- **Time-consuming** (≥ 1 hour per day) or cause clinically significant **distress** or **impairment** in functioning

SPECIFIC PHOBIAS

- Lifetime prevalence: **~10%**
- **F > M**

DSM-5 Criteria:

- Significant **fear/anxiety** about **specific object/situation** (e.g.: heights, animals, blood, etc.)
- In children, fears may express as **crying, tantrums, freezing, or clinging**
- The object/situation **provokes immediate fear/anxiety**
- The object/situation is **actively avoided**
- Fear/anxiety **out of proportion** to the actual threat or sociocultural context
- Persistent fear/anxiety and/or avoidance **≥ 6 months**
- Causes **distress/impairment** in functioning

SELECTIVE MUTISM

- **Failure to speak** in *specific social situations* despite being able to speak in other situations
- Interferes with function
- **≥ 1 month**
- Not due to a lack of knowledge, a language barrier, or a communication disorder

SOCIAL ANXIETY DISORDER (SAD)

DSM-5 Criteria:

- Marked fear/anxiety of **≥ 1 social situations** for **≥ 6 months**
- For children, must occur in **peer settings**
- Fear of being **negatively evaluated** or **humiliated**
- Social situations almost always **induce fear** or **anxiety**
- ***Avoidance*** behaviours
- Fear/anxiety **out of proportion to the actual threat** or sociocultural context
- Causes clinically significant **distress/ impairment** in functioning

ANXIETY MANAGEMENT

- **Psychoeducation** for all patients & families
- Phobias: systematic **de-sensitization**
- GAD, SAD, OCD, selective mutism: **cognitive behavioural therapy (CBT)** +/- selective serotonin reuptake inhibitor (SSRI)

SSRIs

- Sertraline
- Fluoxetine
- Citalopram
- Escitalopram
- Fluvoxamine

Medications: **START LOW, GO SLOW!**

Common side effects: nausea, sleep disturbance, headache, sexual dysfunction in older adolescents, bowel disturbance, tremor, agitation, appetite change, weight gain, etc. It is **important to educate** patients and their families about the potential side effects.

SEROTONIN SYNDROME

Usually, occurs *within hours* of starting a new drug or after a dose increase.

Overdose with SSRIs → **excessive accumulation of serotonin** → serotonin syndrome

- Onset: abrupt
- Neuromuscular findings: myoclonus & tremor
- Reflexes: increased
- Pupils: mydriasis

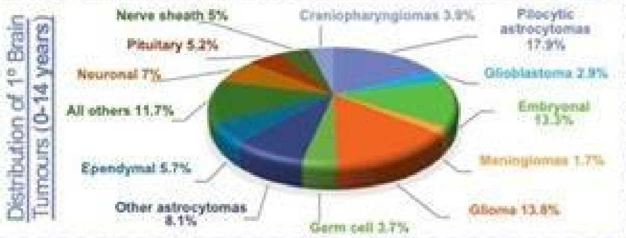
Shivering
Hyperreflexia/myoclonus
Increased temperature
Vital sign instability (↑BP, ↑HR)
Encephalopathy (agitation, delirium, obtundation)
Restlessness/ incoordination
Sweating

June 2020
Katharine V. Jensen (Medical Student 2021, University of Alberta) & Dr. Heidi Wilkes (Pediatric Psychiatrist, University of Alberta) for www.pedscases.com

PEDIATRIC BRAIN TUMOURS

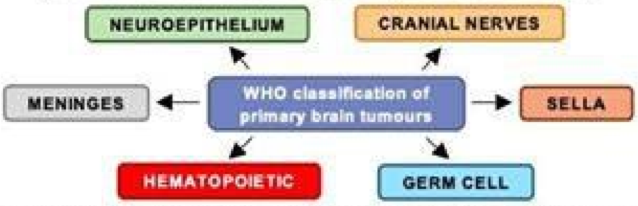
BACKGROUND

- Although rare, brain tumours are the **leading cause of cancer related death** in children.
- Childhood brain tumours have the **second highest incidence** of any pediatric cancer after leukemia. Among childhood solid tumours, brain tumours are the **most common**.
- It is crucial for clinicians to appreciate the signs and symptoms of brain tumours in children, as a **delayed diagnosis can worsen prognosis**.



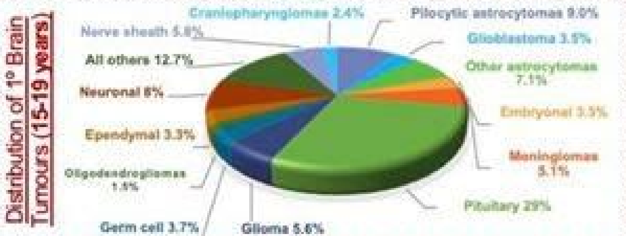
Distribution of 1° Brain Tumours (0-14 years)

Brain tumours are classified according to the **cell of origin**:



WHO classification of primary brain tumours

- NEUROEPITHELIUM
- CRANIAL NERVES
- MENINGES
- SELLA
- HEMATOPOIETIC
- GERM CELL



Distribution of 1° Brain Tumours (15-19 years)

SIGNS & SYMPTOMS

Symptoms are **diverse** and arise based on which **neuroanatomic pathway is disrupted** by the tumour.

Common presenting triad:

Headache
Nausea/ vomiting
Gait imbalance

- Symptoms develop **progressively** over **weeks** to **months**
- Slow steady decline in school performance

The above triad may suggest **cerebrospinal fluid obstruction** secondary to a brain mass.

SIGNS & SYMPTOMS	TUMOUR LOCATION
Recurrent vomiting including early morning; head tilt; enlarged head	Posterior fossa Ventricular system
Visual changes (double vision, vision loss); nystagmus; cranial nerve palsy	Optic pathway, suprasellar, brainstem, posterior fossa
Delayed puberty, anorexia, failure to thrive	Hypothalamic-pituitary axis
Early handedness (< 1 years old)	Cortex, subcortical, brainstem, spinal cord
Tics, tremor, movement disorder	Basal ganglia, thalamus, midbrain

NEURO EXAM	FINDINGS SUGGESTIVE OF A BRAIN TUMOUR
MENTAL STATUS	Progressive neurocognitive decline, encephalopathic
GAIT	Wide-based, unsteady, unable to walk in tandem straight-line
CN II	Visual field deficits, papilledema
CN III, IV, VI	Abnormal extraocular movements, nystagmus (particularly upgaze), gaze paralysis, poorly reactive pupils
CN VIII	Hearing deficits, vertigo
CN IX, X, XII	Dysphagia, drooling
MOTOR EXAM	Early handedness, delayed motor milestones, pronator drift, muscle atrophy associated with focal changes in tone
REFLEXES	Hyperreflexia, positive Babinski sign
COORDINATION	Dysmetria, overshoot on mirror testing, marked asymmetry of finger and/or toe tapping
SENSORY	Focal sensory deficits depending on tumour location

The majority of children with brain tumours will have an **abnormal neurologic exam.**

LATE EFFECTS FROM CANCER THERAPY

- Learning disability, memory problems, lower IQ
- 2° neoplasms
- Endocrinopathy (GH deficiency, precocious puberty, delayed puberty, hypothyroidism)
- Depression/anxiety
- Epilepsy
- Auditory impairment
- Cataracts

INVESTIGATIONS

Neuroimaging: **MRI** of the **brain** and **spine** with and without gadolinium

Preoperative laboratory tests: **CBC, electrolytes, creatinine, coagulation studies, blood type** and **cross-matching**

Other investigations to consider:

- **Endocrine laboratory tests:** growth hormone (GH), prolactin, LH, FSH, cortisol, ACTH, and TSH.
- **Ophthalmologic exam:** establish baseline visual field deficits prior to surgery.
- **Tumour markers:** alpha fetoprotein (AFP) and beta-human chorionic gonadotropin (β-HCG).

MANAGEMENT STRATEGY

- **ABCs** – airway, breathing, circulation
- Multidisciplinary approach: **neurosurgery**, **pediatric oncology**, social work, child life specialists, and neuropsychologists
- Intravenous **steroids** with acid suppression
- Consider **seizure prophylaxis** for patients at high risk for seizures
- While in the ED, the patient can be worked up for any concurrent illnesses
- **Neurosurgery** – maximal safe surgical resection of the tumour
- **Chemotherapy** and **radiation therapy**

April 2020
Katharine V. Jensen (Medical Student 2021, University of Alberta) &
Dr. Bev Wilson (Professor and Pediatric Oncologist, University of Alberta) for www.pedscases.com

PEDIATRIC EPILEPSY

- **Epilepsy** is one of the **most common** chronic neurological disorders. It is characterized by **recurrent unprovoked seizures** or an **enduring predisposition** to generate epileptic seizures. If epilepsy begins in childhood, it is **often outgrown**. Seizures are **common** in childhood and adolescence. Approximately **3%** of children will experience a seizure.
- A seizure occurs when there is a **sudden change in behavior** or **sensation** caused by **abnormal** and **excessive electrical hypersynchronization** of neuronal networks in the cerebral cortex. Normal inhibition is overcome by excessive excitatory stimuli.
- If the cause of the seizures is known (for example: genetic, inborn errors of metabolism, metabolic (eg: low glucose, electrolyte abnormalities), structural (eg: malformations, tumours, bleeds, stroke, traumatic brain injury), infectious, inflammatory, or toxins) it is classified as **symptomatic**. If the cause is unknown, it is classified as **idiopathic**.

1. WHERE DID THE SEIZURE START? / WHAT KIND OF SEIZURE IS IT?

FOCAL ONSET
Seizure that **originates in a focal cortical area** with associated clinical features.

GENERALIZED ONSET
Seizure that involves **both sides of the brain** from the onset.

UNKNOWN ONSET
When it is **unclear** where the seizure starts.

2. IS AWARENESS IMPAIRED?
YES
NO

3. PROGRESSION TO BILATERAL?
YES
NO

SEIZURE SEMIOLOGY (The terminology for seizure types is designed to be useful for communicating the key characteristics of seizures)

CLONIC: sustained rhythmical jerking movements.

TONIC: muscles stiffen or tense.

ATONIC: sudden loss of muscle tone, lasting seconds.

MYOCLONUS: sudden lighting-like jerk, may cluster.

EPILEPTIC SPASM: sudden flexion, extension, or flexion-extension of proximal and truncal muscles (1-2 seconds).

AUTONOMIC: eg: rising epigastric sensations, waves of hot or cold, rapid heart rate, etc.

AUTOMATISMS: stereotyped, purposeless movements. Eg: lip smacking, chewing, finger rolling, picking at clothes, etc.

ABSENCE: brief (≤ 10s), frequent (up to 100's) staring spells, associated with cessation of activity or impaired awareness.

OTHERS: change in cognition, emotions, or sensation. Behavior arrest.

CHILDHOOD EPILEPSY SYNDROMES

Infantile Spasms (IS): epileptic spasms lasting seconds, that occur in clusters on awakening in infants < 1 year. West Syndrome Triad: IS, developmental regression/plateau, hypsarrhythmia on EEG. Care by neurologist required.

Juvenile Myoclonic Epilepsy: onset in puberty of myoclonic jerks, with early morning predominance. +/- generalized tonic-clonic and/or absence seizures.
Tx: valproic acid, lamotrigine, levetiracetam.

Childhood Absence Epilepsy: brief (≤ 10 secs), frequent (up to 100's) staring spells with impaired awareness, provoked by hyperventilation (can test clinically). No post-ictal period. EEG pattern of 3 Hz. Peak age 5-6 years old, often outgrown. May be inherited. DDx: behavioral staring spells, inattentiveness, and focal seizures with impaired awareness. *Tx: ethosuximide, valproic acid, lamotrigine.*

Rolandic Epilepsy: infrequent, focal onset seizures arising from the Rolandic cortex, commonly occurring nocturnally or upon awakening. Episodes involve twitching or stiffness in the face. Seizures may not require treatment and generally cease by mid/late adolescence. *Tx: levetiracetam, carbamazepine, clobazam.*

Lennox-Gastaut Syndrome: an epileptic encephalopathy, characterized by several different seizure types (tonic, atonic, generalized tonic-clonic, absence (atypical), etc.) that are often refractory. Seizure onset typically between 1-7 years but may be preceded by infantile spasms. Structural brain abnormalities and genetic etiologies are common. *Tx: valproic acid, clobazam, stiripentol, rufinamide.*

Childhood Occipital Epilepsy: Gastaut type: brief, frequent focal sensory visual seizures with onset from 18 months to 19 years. Remission in 50-60%.
Tx: carbamazepine. Panayiotopoulos syndrome: infrequent focal prolonged autonomic seizures, onset in early childhood. Predominant symptom is prolonged episodes of nausea and vomiting. EEG: focal, high amplitude spikes over the occipital regions. Self-limiting. *Tx: carbamazepine.*

Landau-Kleffner Syndrome: rare disorder of acquired receptive aphasia and agnosia in children ≤ 6 years old. Abnormal EEG with continuous spike-waves during slow sleep (CSWS). Convulsive, non-convulsive seizures, or no seizures at all. *Tx: high dose diazepam.*

Reflex Epilepsy: a specific stimulus (visual, auditory, etc.) or event triggers seizures (eg: photosensitive epilepsy in response to lights flickering). Absence of spontaneous seizures. *Tx: valproic acid, clonazepam, clobazam, lamotrigine.*

⚠ SEIZURE SAFETY ⚠

Educate patient/family about risks around water (swimming, bathing), fire/cooking, heights, and prolonged seizures. No driving until cleared by an MD.

DIAGNOSIS & MANAGEMENT

Medical history, including family history.
Seizure description: pre-ictal, ictal, and post-ictal.
Physical exam: thorough neurologic and mental status evaluation (eg: vital signs, response testing, memory recall testing, pupillary response, etc.).

↓

Diagnostic tests:
- Electroencephalogram (EEG) – records the brain's electrical activity.
- Neuroimaging – CT or MRI to provide structural information.

↓

COMMON ANTICONVULSANTS: ativan, carbamazepine, clobazam, clonazepam, ethosuximide, lamotrigine, levetiracetam, phenobarbital, phenytoin, valproic acid

↓

Surgical evaluation is considered in **medically refractory** (failed 2 appropriately chosen and dosed medications) or **intractable** epilepsies.

↓

Ketogenic diet: high in fats; low in protein and carbohydrates; may be considered in difficult to control epilepsy.

March 2020
Katharine V. Jensen (Medical Student 2021, University of Alberta) &
Dr. Natarie Liu (Neurologist and Epileptologist, University of Alberta) for www.pedscases.com

PEDIATRIC HISTORY (HEADS)

CREATE A SAFE ENVIRONMENT

- Set **clear expectations** with patient and parents.

  It is typical to first talk 1-on-1 with the child, then 1-on-1 with the parents, and bring them all together at the end.
- Review **confidentiality** and encourage participation.

  "Everything will be confidential between us meaning I can't share any information you don't want me to EXCEPT the following reasons: (1) you are at risk of harming yourself, (2) someone is harming you, or (3) you are at risk of harming someone else."

ASK OPEN-ENDED QUESTIONS TO BUILD RAPPORT

- Invite patient to describe **chief concern** and discuss **history of present illness**.
- Consider using the **HEADS framework** to build rapport and obtain relevant psychosocial history.

  Home Life: "Who lives with you?" / "How do you get along with your mom and/or dad?" / "How do they get along?" / "How is your relationship with your siblings?" / "What things do you do together?"

  Education: "Are you in school?" / "Which grade are you in?" / "How do you spend time in recess?" / "Do you have anyone in school whom you consider as a friend?" / "What is your favorite subject?" / "How do you feel about your report card?" / "What do you want to do when you finish school?"

  Eating: "What do you usually have for breakfast, lunch and dinner?" / "Do you have fruits, vegetables, whole grains, and proteins every meal?" / "Do you have any food restrictions?" / "What does a healthy diet look like to you?"

  Activities: "What do you do for fun?" / "How do you spend your free time?" / "Are you in any sports or activities?" / "How do you get along with other people?"

  Drug / Substance Use: "Many young people experiment with drugs, alcohol or cigarettes. Have you ever tried them to relax or get high? What have you tried?" / "Do any of your friends take drugs?" / "Are there any drugs at your school?"

  Depression - Ask all patients about their mood: "On most days, how do you feel?" / "How often do you feel this way?" / "When did this feeling begin?" / "How long have you had this feeling?"

  Suicidality & Safety: "Do you have any dark thoughts about harming yourself or suicide?" / "Do you want to disappear or run away?" / "How long have you had these thoughts?" / "Have those thoughts ever gotten so bad that you acted on them?" / "Do you have any plan to harm yourself or others?"

  Sexuality: "How do you describe your sexual orientation?" / "How do you identify your gender?" / "Are you or have you been involved in a relationship?" / "How was the relationship?" / "Have you been sexually active?"
- Remember other **social or behavioral factors** may also contribute to the chief concern (e.g., employment, exercise, spirituality, housing status).
- The Canadian Pediatric Society website (www.cps.ca) has a list of validated screening tools and rating scales for suspected mental health conditions.

September 2022
Annabelle Wong (Medical Student, University of Alberta) and
Dr. Heidi Wilkes (Consulting Psychiatrist, Stollery Children's Hospital) for www.pedscases.com

PEDIATRIC HYPOCALCEMIA

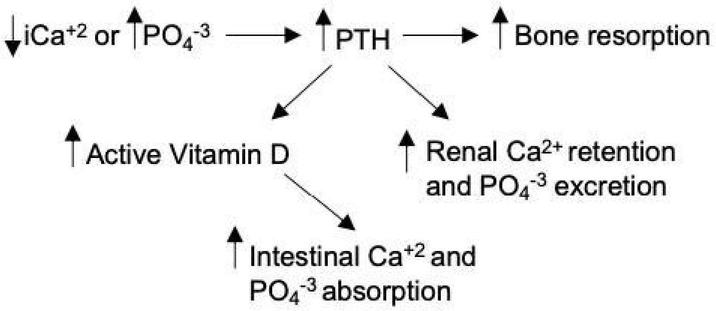
DEFINITION: low *ionized* calcium

Causes divided based on PTH response:

Inappropriately low PTH: Congenital or acquired hypoparathyroidism

Appropriately high PTH:
- **Vitamin-D related:** Deficiency or disorders of vitamin D metabolism
- **Non-vitamin D related:** Kidney disease, increased phosphate (i.e., enemas, tumor lysis), PTH resistance (i.e., pseudohypoparathyroidism)

CALCIUM METABOLISM



↓iCa^{+2} or ↑PO_4^{-3} → ↑PTH → ↑Bone resorption

↑Active Vitamin D

↑Renal Ca^{2+} retention and PO_4^{-3} excretion

↑Intestinal Ca^{+2} and PO_4^{-3} absorption

Hypocalcemia stimulates PTH causing Vitamin D activation, increased Ca^{+2} and net decreased PO_4^{-3}

PRESENTATION

SYMPTOMS	SIGNS
Infants: Muscle spasm Feeding difficulties Jitteriness	**Chvostek's:** *Facial muscle contraction* elicited by tapping over the ipsilateral facial nerve
Children: Muscle spasm Paresthesia Numbness	**Trousseau's:** *Carpal spasm* following inflation of a blood pressure cuff to above systolic for 3 mins.
Life-threatening: Laryngospasm Seizure Arrythmia (i.e., QT prolongation)	

INVESTIGATIONS

- PTH, Vitamin D, Ca^{+2}, PO_4^{-3}, Mg^{+2}
- Urinary Ca, spot Ca:Cr ratio, blood gas, albumin
- **Caution:** Both Ca^{+2} and H^+ bind albumin. Thus, alkalosis decreases iCa^{+2} causing true hypocalcemia.
- Hypoalbuminemia results in *pseudohypocalcemia*, as total Ca^{+2} is low but iCa^{+2} is normal. This is confirmed with a correction formula.

DIAGNOSIS

Diagnosis	PTH	Ca^{+2}	PO_4^{-3}	Vit D
Hypoparathyroidism	↓	↓	↑	WNL
Vit D deficiency	↑	WNL or ↓	WNL or ↓	↓
PTH resistance	↑	↓	↑	WNL

MANAGEMENT

Acute Symptomatic	Acute Asymptomatic	Long-term Management
IV calcium bolus if tetany, seizures, QT prolongation, or laryngospasm **Arrythmia alert:** Cardiac monitoring is required during IV Ca^{+2} infusion	Oral therapy as per long-term management if asymptomatic or mild symptoms, such as paresthesia	**Hypoparathyroidism or PTH resistance:** Ca^{2+} and activated vitamin D supplements **Vitamin D deficiency:** Inactive vitamin D supplement **Renal failure:** Phosphate binders, activated vitamin D

August 2022
Dr. Madeline Parker (Resident, University of Saskatchewan), Dr. Munier Nour (Pediatric Endocrinologist, University of Saskatchewan) for www.pedscases.com

PEDIATRIC MIGRAINE

PEDIATRIC MIGRAINE CRITERIA (International Classification of Headache Disorders, 3rd Edition)

MIGRAINE WITHOUT AURA

60-80% of childhood migraines

A. At least **5 distinct attacks** meeting the following criteria
B. Attacks lasting **1*-72 hours**
C. At least 2 of the following:
  - **Bilateral*** or **unilateral**
  - **Pulsing** or **throbbing** quality
  - **Moderate** or **severe pain** intensity
  - Aggravated by or causing avoidance of **physical activity**
D. At least one of the following during the headache:
  - **Nausea** and/or **vomiting**
  - **Photophobia** and **phonophobia**
E. Not attributed to another diagnosis

** Different from adult migraine*

MIGRAINE WITH AURA

15-30% of childhood migraines

A. At least **2 distinct attacks** meeting the following criteria
B. Aura consisting of at least one of the following reversible symptoms:
  - **Visual** *(most common)*, **sensory, dysphasic speech disturbance, motor, brainstem,** and/or **retinal symptoms**
C. At least 3 of the following:
  - At least 1 aura symptom **develops gradually over ≥ 5 minutes**
  - 2 or more aura symptoms occur in **succession**
  - Each aura symptom lasts **≥ 5** and **≤ 60 minutes**
  - At least 1 aura symptom is **unilateral**
  - At least 1 aura symptom is **positive** (scintillations, pins & needles, etc)
  - Headache begins **during aura** or **follows the aura within 60 minutes**
D. Not attributed to another diagnosis

ASSOCIATED CONDITIONS

Episodic syndromes:
- Cyclic vomiting syndrome
- Abdominal migraine
- Benign paroxysmal vertigo
- Benign paroxysmal torticollis

CHRONIC MIGRAINE

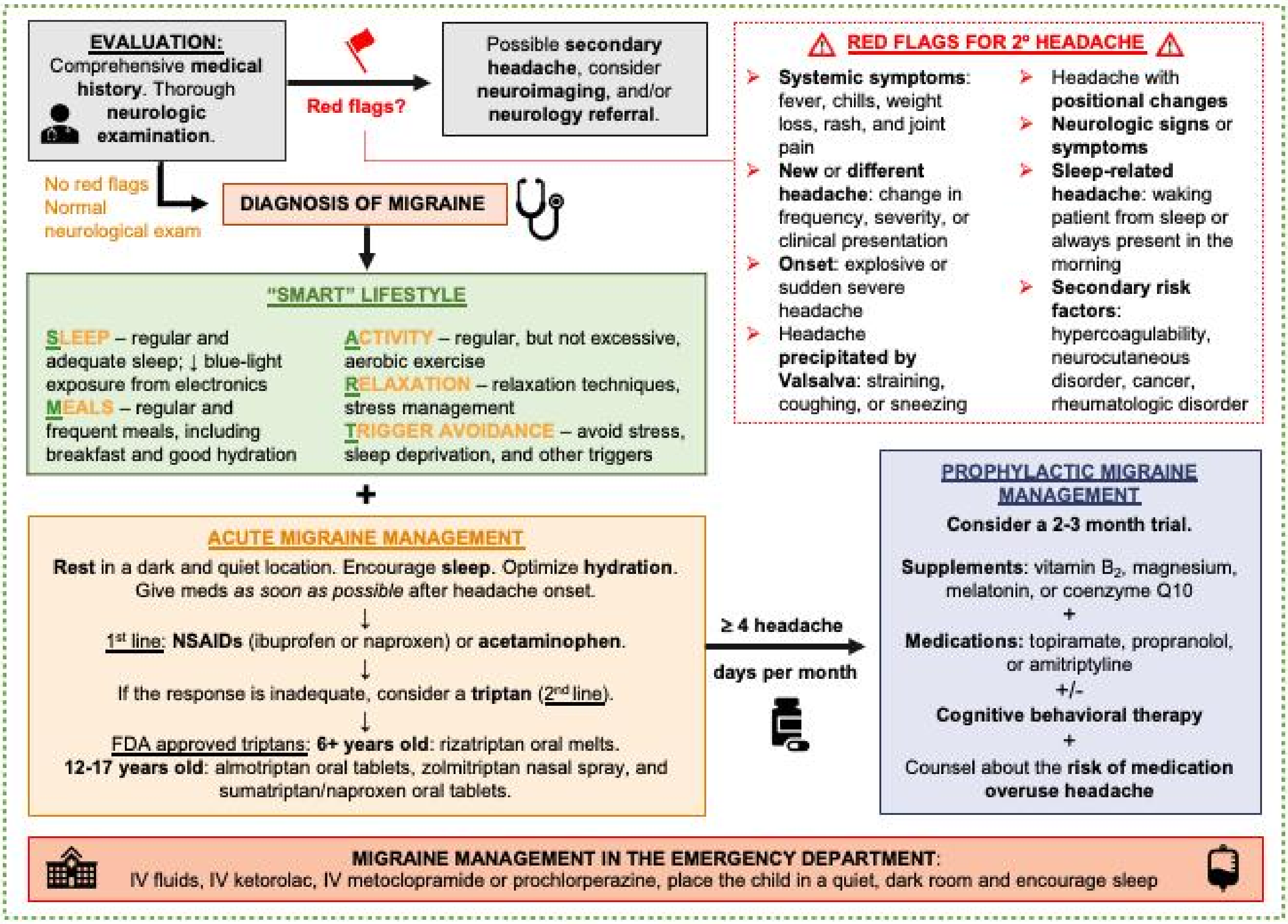
- Headache occurring on **≥ 15 days/month** for **≥ 3 months.**
- Patient has features of **migraine headache** on at least **8 days/month.**

MEDICATION OVERUSE HEADACHE (MOH)

- Worsening headache that develops in a patient with **pre-existing primary headache,** who uses **acute headache medication ≥ 15 days/month** for **3 months.**
- 50% of patients diagnosed with chronic migraine have MOH, which returns to an episodic migraine once the offending medication is discontinued.

STATUS MIGRAINOSUS

- A migraine that is **unremitting for ≥ 72 hours** with **debilitating pain** and/or associated symptoms.
- Often associated with medication overuse.



EVALUATION: Comprehensive **medical history**. Thorough **neurologic examination.**

Red flags? → Possible **secondary headache**, consider **neuroimaging**, and/or **neurology referral**.

No red flags
Normal neurological exam → **DIAGNOSIS OF MIGRAINE**

⚠ RED FLAGS FOR 2° HEADACHE ⚠

- **Systemic symptoms**: fever, chills, weight loss, rash, and joint pain
- **New** or **different headache**: change in frequency, severity, or clinical presentation
- **Onset**: explosive or sudden severe headache
- Headache **precipitated by Valsalva**: straining, coughing, or sneezing
- Headache with **positional changes**
- **Neurologic signs** or **symptoms**
- **Sleep-related headache**: waking patient from sleep or always present in the morning
- **Secondary risk factors:** hypercoagulability, neurocutaneous disorder, cancer, rheumatologic disorder

"SMART" LIFESTYLE

SLEEP – regular and adequate sleep; ↓ blue-light exposure from electronics
MEALS – regular and frequent meals, including breakfast and good hydration
ACTIVITY – regular, but not excessive, aerobic exercise
RELAXATION – relaxation techniques, stress management
TRIGGER AVOIDANCE – avoid stress, sleep deprivation, and other triggers

+

ACUTE MIGRAINE MANAGEMENT

Rest in a dark and quiet location. Encourage **sleep**. Optimize **hydration**. Give meds *as soon as possible* after headache onset.

↓

1st line: **NSAIDs** (ibuprofen or naproxen) or **acetaminophen**.

↓

If the response is inadequate, consider a **triptan** (2nd line).

↓

FDA approved triptans: **6+ years old**: rizatriptan oral melts.
12-17 years old: almotriptan oral tablets, zolmitriptan nasal spray, and sumatriptan/naproxen oral tablets.

≥ 4 headache days per month →

PROPHYLACTIC MIGRAINE MANAGEMENT

Consider a 2-3 month trial.

Supplements: vitamin B_2, magnesium, melatonin, or coenzyme Q10
+
Medications: topiramate, propranolol, or amitriptyline
+/-
Cognitive behavioral therapy
+
Counsel about the **risk of medication overuse headache**

MIGRAINE MANAGEMENT IN THE EMERGENCY DEPARTMENT:

IV fluids, IV ketorolac, IV metoclopramide or prochlorperazine, place the child in a quiet, dark room and encourage sleep

February 2020
Katharine V. Jensen (Medical Student 2021, University of Alberta) &
Dr. Colin Wilbur (Pediatric Neurologist and Assistant Professor, University of Alberta) for www.pedscases.com

PEDIATRIC PRESCRIPTION WRITING

Include multiple **PATIENT IDENTIFIERS** (name, date of birth, health care #, address)

MEDICATION:

name of prescribed medication and form (capsule, tablet, liquid)

ROUTE:

specify how the medication is to be given: orally (PO), nasogastric (NG), G-tube, rectal (PR), intranasal, inhaled, intra-muscular (IM), intra-venous (IV), subcutaneous (SC), topical.

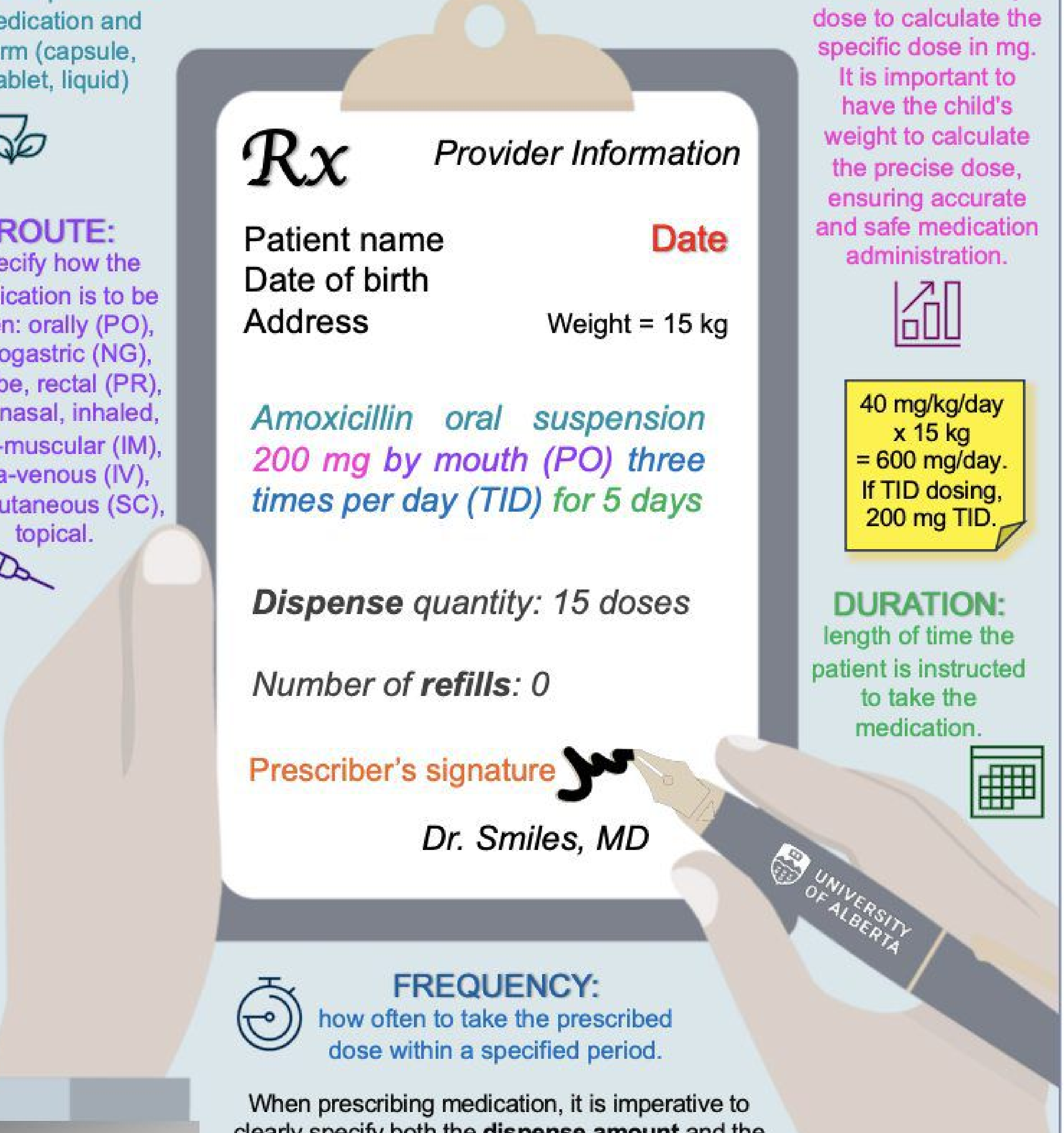
DOSAGE:

use the total daily dose to calculate the specific dose in mg. It is important to have the child's weight to calculate the precise dose, ensuring accurate and safe medication administration.

40 mg/kg/day
x 15 kg
= 600 mg/day.
If TID dosing,
200 mg TID.

DURATION:

length of time the patient is instructed to take the medication.



Rx *Provider Information*

Patient name
Date of birth
Address

Date

Weight = 15 kg

Amoxicillin oral suspension 200 mg by mouth (PO) three times per day (TID) for 5 days

***Dispense** quantity: 15 doses*

*Number of **refills**: 0*

Prescriber's signature

Dr. Smiles, MD

FREQUENCY:

how often to take the prescribed dose within a specified period.

When prescribing medication, it is imperative to clearly specify both the **dispense amount** and the **number of refills** to ensure accurate dispensing and provide clear guidance for treatment plan.

March 2024
Dr. Katharine V. Jensen (Pediatrics Resident, University of Alberta) and Dr. Karen Forbes (Professor of Pediatrics, University of Alberta) for www.pedscases.com

PEDIATRIC VITAL SIGNS REFERENCE CHART

Heart Rate (beats/min)

Age	Awake	Asleep
Neonate (<28 d)	100-205	90-160
Infant (1-12 mos)	100-190	
Toddler (1-2 y)	98-140	80-120
Preschool (3-5 y)	80-120	65-100
School-age (6-11 y)	75-118	58-90
Adolescent (12-15 y)	60-100	50-90

Respiratory Rate (breaths/min)

Age	Normal
Infant (<1 y)	30-53
Toddler (1-2 y)	22-37
Preschool (3-5 y)	20-28
School-age (6-11 y)	18-25
Adolescent (12-15 y)	12-20

Reference: PALS Guidelines, 2015

Blood Pressure (mmHg)

Age		Systolic	Diastolic	Systolic Hypotension
Birth (12 h)	<1 kg	39-59	16-36	<40-50
	3 kg	60-76	31-45	<50
Neonate (96 h)		67-84	35-53	<60
Infant (1-12 mos)		72-104	37-56	<70
Toddler (1-2 y)		86-106	42-63	<70 + (age in years × 2)
Preschool (3-5 y)		89-112	46-72	
School-age (6-9 y)		97-115	57-76	
Preadolescent (10-11 y)		102-120	61-80	<90
Adolescent (12-15 y)		110-131	64-83	

Reference: PALS Guidelines, 2015
For diagnosis of hypertension, refer to the 2017 AAP guidelines Table 4 & 5:
http://pediatrics.aappublications.org/content/early/2017/08/21/peds.2017-1904

Temperature (°C)

Method	Normal
Rectal	36.6-38.0
Tympanic	35.8-38.0
Oral	35.5-37.5
Axillary	36.5-37.5

Ranges do not vary with age.
Screening: axillary, temporal, tympanic (↓ accuracy)
Definitive: rectal & oral (↑ reflection of core temp.)
Reference: CPS Position Statement on Temperature Measurement in Pediatrics (2015)

Oxygen Saturation (SpO_2)

SpO_2 is lower in the immediate newborn period. Beyond this period, a SpO_2 of **<90-92%** may suggest a **respiratory condition** or **cyanotic heart disease**.

Dr. Chris Novak & Dr. Peter Gill for www.pedscases.com
(Edited March 2020 by Richard He)

PERIORIFICIAL DERMATITIS (POD)

Persistent, inflammatory, papular facial rash commonly affecting the perioral area, thought to be related to **deficiencies in skin barrier function**.

EPIDEMIOLOGY

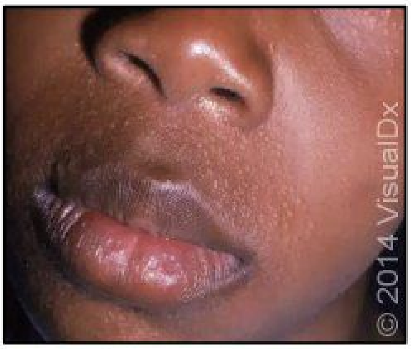
- Average age of onset **6-7 years**, can be seen as young as 3 months
- Most common in **Fitzpatrick phototypes I-II**
- **Granulomatous variant** more common in **Fitzpatrick phototypes IV-VI**

PRESENTATION

- Classically seen in a child using **topical corticosteroids** (TCS) or immediately following a period of facial TCS use (Steroid Rebound Effect)
- Largely **asymptomatic**, may be mildly pruritic, burning, or tender
- Parents may suspect acne, especially in teens

PHYSICAL EXAM

- Monomorphic, 1-2 mm pink papules, fine pustules, eczematous patches, and thin plaques
- Granulomatous variant: skin-colored, hyperpigmented, or dull pink-red papules
- Typically, around mouth, sparing the vermillion border, nose, and eyes



DIAGNOSIS

Diagnosis made **clinically** based on:

- **Characteristic appearance** and **distribution** of rash
- **Supportive history** (aggravating factors identified)

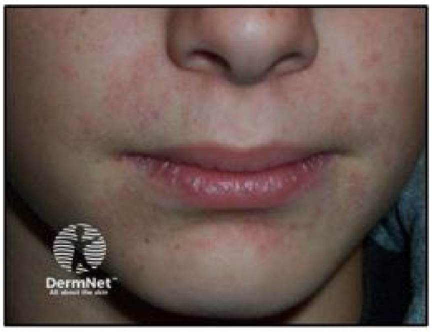
Other investigations (swabs, skin scrapings, biopsies) rarely necessary.

MANAGEMENT

- **Initial approach is conservative**
- Identify and discontinue **aggravating factors**
- Consider **tapering facial TCS** to reduce risk of rebound flare
- **POD is self-limiting when offending agent(s) removed**

If bothersome or distressing, consider further medical management in addition to the above:

Mild: Topical Therapies	**Moderate, Severe, or Refractory: Oral Antibiotics**
Pimecrolimus 1% cream	≥ 8 years: Tetracycline PO
Metronidazole 1% cream	< 8 years: Macrolide PO
Erythromycin 1% gel	



COMMON TRIGGERS

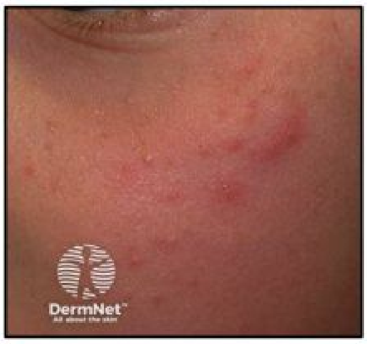
- Topical, oral, inhaled, or nebulized **corticosteroids**
- Fluoridated toothpastes and oral rinses
- Occlusive facial products (creams, sunscreens, cosmetics, face masks)

DIFFERENTIAL Dx

- Acne Vulgaris
- Rosacea
- Contact dermatitis
- Seborrheic dermatitis
- Impetigo
- Dermatophyte infections
- Demodex folliculitis

PROGNOSIS

- Benign, generally non-scarring
- Can take several weeks to months to resolve
- Pigmentation changes may persist



January 2025
Lauren Balogh (MD/MBA Student, University of Alberta), Dr. Loretta Fiorillo (Pediatric Dermatology, University of Alberta) for www.pedscases.com

Images obtained with permission from DermNet (Right) and Skinsight (Left). Left image displays Granulomatous POD.

PNEUMOTHORAX

A pneumothorax is an accumulation of air within the pleural space, the space between the chest wall and lung tissue. There are two main categories of pneumothorax, **traumatic** and **spontaneous**. Traumatic pneumothorax can be broken into **accidental** and **iatrogenic**, and spontaneous pneumothorax can be broken down into **primary** (otherwise healthy lung) or **secondary** (underlying lung conditions). Spontaneous pneumothoraxes are rare, occurring in 2.6/100,000 children.

DIFFERENTIAL DIAGNOSES

These conditions can partially mimic or mask a pneumothorax.

Pulmonary embolism
- ☐ History of DVT
- ☐ Swollen/tender leg
- ☐ Long periods of immobility

Myocardial infarction
- ☐ Rare in pediatrics
- ☐ Radiation of chest pain
- ☐ Pain on exertion
- ☐ Past history of cardiac conditions
- ☐ Congenital/acquired heart conditions

Esophageal rupture
- ☐ Extreme retrosternal chest pain
- ☐ Severe vomiting/retching

MSK-type pain
- ☐ Pain on palpation

CLINICAL PRESENTATION

- Sudden, sharp pain on the lateral side of the body, localizing to one side
- May complain of cough or palpitations
- Lightheadedness (from hypotension, if present)
- Primary spontaneous pneumothorax: intense pain at onset, then dissipate within 24h
- Secondary/traumatic pneumothorax: more severe pain that does not go away

PNEUMOTHORAX IN INFANTS

In infants, the signs are slightly different. Look for:
- Cyanosis
- Nasal Flaring
- Tachypnea
- Decreased breath sounds
- Chest transillumination

INVESTIGATIONS

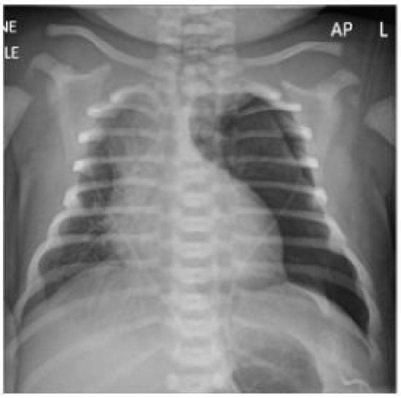
- CXR to confirm clinical suspicion: standing PA with inspiration/expiration views
- ABG (may be normal in acute setting)
- ECG to rule out cardiac cause

PHYSICAL EXAM

Vital Signs
- Tachypnea
- Tachycardia

Respiratory Exam
- ↓/absent breath sounds on the side of the affected lung
- Transmitted breath sounds
- ↓ chest movement with breathing
- ↓ tactile fremitus
- Enlarged hemithorax (asymmetry of the chest wall)
- Labored breathing
- Hyperresonance to percussion on affected side
- May have associated subcutaneous emphysema

Watch for signs of **tension pneumothorax**, including tracheal deviation, sudden difficulty ventilating, hypotension, distended neck veins, or an elevated JVP.



MANAGEMENT

All patients, regardless of treatment, should have a follow-up CXR (timing of which depends on clinical status).

For an asymptomatic patient with a:
1. Spontaneous pneumothorax <3cm between apex of lung and dome of thoracic cavity OR
2. Pneumothorax involving <20% of the hemithorax

Observation alone is a satisfactory treatment.

For other conditions including:
1. A large primary spontaneous pneumothorax OR
2. A tension pneumothorax

Patients are treated with a **tube thoracostomy.**

If recurrent, bilateral, or persistent air leak, a thoracotomy, or video-assisted thoracoscopic surgery, may be necessary.

Published July 2022
Morgan Gregg (Medical Student, University of Alberta) and Dr. Foulds (Assistant Professor, Division of Pediatric Hospital Medicine, University of Alberta) for www.pedscases.com

PROBIOTIC USE IN PEDIATRICS

Probiotics:
Live microorganisms that when consumed in **adequate** amounts, alter the host microflora and confer a health **benefit**.

Probiotic Properties

- ☐ Able to resist processing and **survive** in the digestive tract
- ☐ Are **non-pathogenic** in a normal host
- ☐ Different strains have **unique** properties

Intestinal Microflora

- ☐ Colonization of the gut begins **immediately** after birth
- ☐ Microflora composition is **influenced by**:
  - ○ Gestational age
  - ○ Delivery type (vaginal, c-section)
  - ○ Antibiotic use
  - ○ Environment
  - ○ Diet (breast milk, formula)
- ☐ Addition of **solid** foods to diet causes the infant gut to resemble that of the **adult**

Probiotics effects on conditions are dependent on the **specific strain** and ensuring an **adequate dose**!

Role of Gut Microflora

- ☐ **Intestinal** microflora support the intestinal mucosa **barrier function** by:
  - ○ Increasing **mucin** secretion
  - ○ Decreasing gut **permeability**
  - ○ Modulating gut **immune** function
  - ○ **Competing** with pathogenic bacteria for adhesion receptors and nutrients
- ☐ **Colonic** microflora **metabolize** malabsorbed carbohydrates into **short chain fatty acids:**
  - ○ These are the enterocyte's **preferred** fuel
  - ○ Also **acidifies** colon contents and increases water **reabsorption**

Probiotics support these gut microflora functions!

Adverse Effects

- ☐ Probiotics have the potential to cause local or invasive **infection** in **any** patient
- ☐ Use with **caution** in those with a higher risk of infection: born **preterm**, immunocompromised

Indication	Strains with the most evidence	Recommendations, definitions, and mechanisms	Evidence of Benefit
Prevention of AAD	LGG S boulardii	AAD (Antibiotic associated diarrhea): ≥3 loose stools/day for ≥2 days, occurring up to 2 weeks after antibiotic initiation	Yes
Treatment of Acute Viral Diarrhea	LGG Strain and dose dependent	Most benefit if started **within 48 hours** of illness **Reduces duration** of diarrhea by 17-30 hours **Rotavirus** is more responsive **No** effect on **bacterial** diarrhea	Yes
Prevention of NEC	Bifidobacterium infantis, B bifidus, S thermophilus	NEC (necrotizing enterocolitis) pathology related to altered gut **permeability and microflora** Promote the use of probiotics with **breastfeeding** in **preterm** infants **>1 kg** who are **at risk of NEC**	Yes
Preventing infectious diarrhea	Strain dependent	More effect in **non**-breastfed infants Consider use in those who: live in **long term care** facilities or attend **daycare** and have **recurrent** infections	Modest
IBS	LGG, Escherichia coli, VSL#3	IBS (irritable bowel syndrome) **symptom improvement**: abdominal distension, gassiness and IBS scores	Possible
Infantile Colic	L reuteri	Colicky infant guts have reduced levels of lactobacilli May reduce **symptoms** of colic	Possible
C difficile infection	S boulardii	Possible role in **preventing** recurrent infections **No** evidence in primary prevention	Possible

More evidence needed **before** these potential indications can be recommended: Reduction of **respiratory infections**, **antibiotic** use and absences from daycare due to illness; treatment of **allergic colitis**; prevention of **traveler's diarrhea**; use in **preterm babies weighing <1 kg**; prevention and treatment of **atopic and allergic** disease.

Reference: Canadian Paediatric Society (2019). ***Using Probiotics in the Paediatric Population.*** **Retrieved from https://cps.ca/en/documents/position/probiotics-in-the-paediatric-population**

August 2022
Eileen Tu (Medical Student, C2023, University of Alberta), Dr. Karen Forbes (Pediatrician, University of Alberta), for www.pedscases.com

PAIN & DISTRESS MANAGEMENT FOR BRIEF MEDICAL PROCEDURES

COMMON PAINFUL BRIEF MEDICAL PROCEDURES

- Intravenous cannulation
- Blood draws
- Heel lances
- Lumbar puncture
- Intramuscular injections
- Urine collection in infants
- Abscess drainage
- Dressings for burns
- Nasogastric tube insertion
- Laceration repair
- X-ray for fractures/dislocations

Choose the most **minimally invasive** approach possible. Always try to **combine preventative, physical, psychological,** and **pharmacological approaches** to pain and distress management.

PREVENTION

Whenever possible:

- Choose venipuncture over heel lances or IM injections.
- Couple IV insertion together with blood tests.
- Daily bloodwork should not be prescribed in an automatic, recurring fashion.
- Group blood tests together.

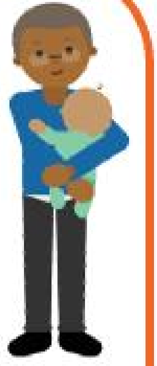
PSYCHOLOGICAL

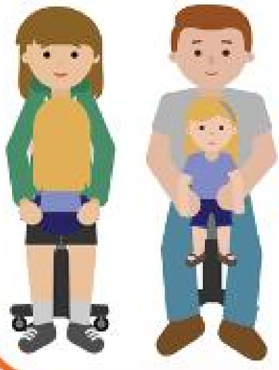
- **Caregiver** presence and involvement
- Use **calm language**
- **Avoid false or premature reassurance**. Don't say things like *"this won't hurt", "it's almost over"*.
- **Age-appropriate preparation**: can show younger children medical supplies to familiarize them with the equipment; for children ≥ 4 years, explain the procedure in a developmentally-appropriate way. Offer them realistic choices.
- **Child-friendly environment**: toys, wall decor, pictures
- **Distraction**:
  - Involve child life therapist, if available
  - Engage the child in non-procedural conversation
  - Bubbles, reading, videos, games, music
  - Deep breathing

PHYSICAL

Infants:

- Pacifier
- Swaddling
- Tucking
- Rocking
- Breastfeeding or sucrose
- Skin-to-skin (kangaroo) care





Children:

- Sitting on caregiver's lap
- Comfort holds
- Sitting upright, rather than lying down, enhances the child's sense of control

PHARMACOLOGICAL

Topical anesthetics:

- Liposomal lidocaine (Maxilene)
- Amethocaine (Ametop)
- Lidocaine-prilocaine (EMLA)
- Vapo-coolant spray (Pain Ease)
- LET gel (lidocaine 4%/ epinephrine 0.1%/ tetracaine 0.5%)

Local anesthetics:

- Lidocaine 1%
- Nerve block

Systemic:

- **Ibuprofen** is superior to acetaminophen for acute pain.
- Ibuprofen can be used for mild to moderate pain or as co-analgesia for moderate to severe pain.
- **Nitrous oxide** – mild sedation & analgesia offered for older children, for some procedures.
- **Intranasal fentanyl** for moderate to severe pain.

Published January 24, 2021
Katharine V. Jensen (Medical Student 2021, University of Alberta), Dr. Evelyne Trottier (Université de Montréal), Dr. Samina Ali (University of Alberta), and Dr. Hayley Turnbull (University of Alberta) for www.pedscases.com. Images provided by ECHO Research, University of Alberta (Dr. Shannon Scott, PI).

PSYCHIATRIC EVALUATION TEMPLATE

Patient ID

Name (Last, First):
DOB: Age: Sex:
Mode of Admission (if applicable):
PHN:
Ethnicity:
Date of Evaluation:

Source of History

Chief Concern

History of Present Illness

Description (***COLDER mnemonic***)
- Characteristics of symptoms
- Onset
- Location / Situation
- Duration
- Exacerbation / Stressor
- Relief

Response
- What makes it better / worse?
- Adaptive skills? Patient assets?

Impairment
- Depression? Suicidal thoughts? Safety?

Relevant Medical History

Past Psychiatric Hx
- Successful & unsuccessful treatments?
- Previous hospitalizations?
- Previous suicide attempts?

Past Medical Hx (Head trauma? Seizure?)
Medications & Allergies
Past Surgical Hx
Birth, Developmental & Behavioral Hx
Targeted Family Hx

Personal & Social History

Home
Education / Employment
Eating & **E**xercise
Activities / Interest
Drugs / Substance Use
Sexuality
Spirituality
Safety / Adverse events

Lab Values / Screening Tools

Height:
Weight:
Tool(s):
Score:

Mental Status Examination

General Appearance
Attire
Grooming / Hygiene
Eye Contact
Attitude (Cooperative?)
Facial Expression
Mood
Affect
Behavior
Motor Activity
Speech
Thought Process & Content
- Alert & Oriented
- Hallucinations
- Delusion(s)

Overall Cognitive Functioning
- Memory
- Age-appropriate knowledge
- Concentration

Insight
Judgment
- Reliability
- Impulse control

Diagnostic Impressions

A summary of the most pertinent features of the examination, including clinician's opinion and impressions about the most likely or relevant diagnoses.

Treatment Plan

Safety
- Is the patient certifiable?
- Should the patient be admitted?
- What level of observation is required?

Medication
Therapy
Investigations
Education / Instructions Given to Patient
Follow Up Plans

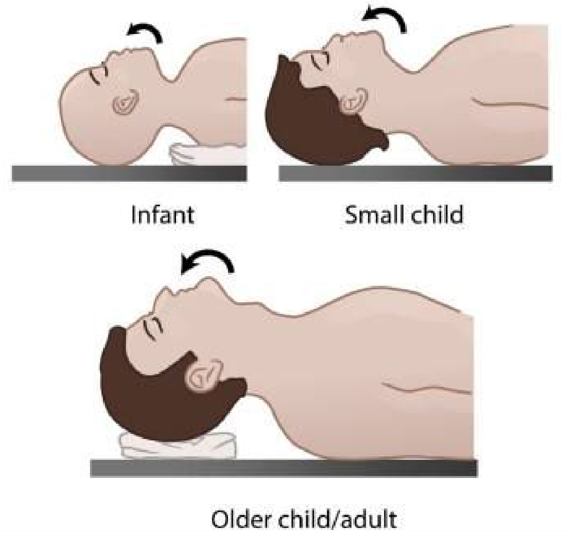
September 2022
Annabelle Wong (Medical Student, University of Alberta) and
Dr. Heidi Wilkes (Consulting Psychiatrist, Stollery Children's Hospital) for www.pedscases.com

RAPID SEQUENCE INTUBATION (RSI)

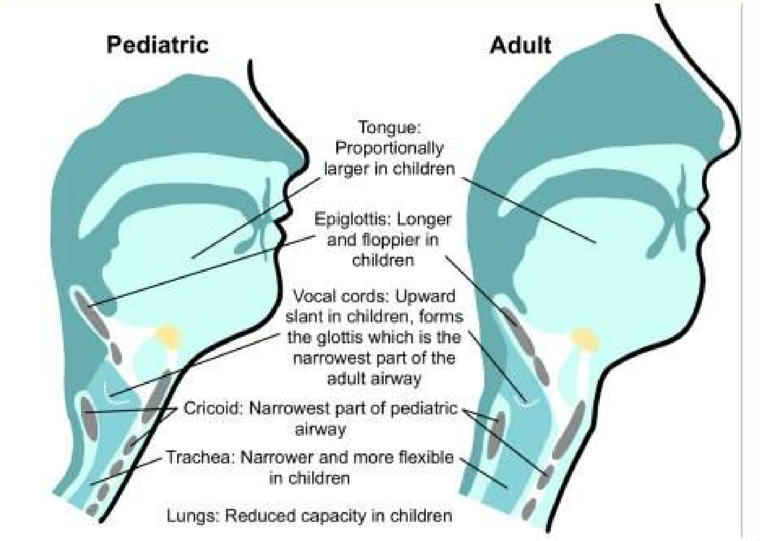
APPROACH TO RSI

1) Preparation	- Identify patient factors that may impact medication choice such as cardiovascular status, raised ICP, bronchospasm, congenital heart defects, or syndromic child - Identify risk factors for difficult airway management including airway trauma, obstruction, and anatomical abnormalities due to genetic syndromes - Assemble and check equipment including monitors, IV, large-bore suction, endotracheal tube (ETT) and laryngoscope and discuss contingency plan
2) Preoxygenation	- Ideally with 100% FiO2 via nonrebreather mask or face mask - Enrich environment with oxygen with a mask nearby patient's head in uncooperative child - Apneic oxygenation via nasal cannula while intubating
3) Pre-treatment	- Atropine (an anticholinergic) to prevent bradycardia is optional and more often used in children <1 year of age - Midazolam (a benzodiazepine) or ketamine (a dissociative anesthetic) in an uncooperative child to facilitate preoxygenation
4) Induction	- Choice of induction agent depends on factors such as hemodynamic stability, septic shock, raised ICP, status asthmaticus, and status epilepticus - Etomidate, propofol, and ketamine are commonly used +/- opioids like fentanyl or remifentanil
5) Paralysis	- Rocuronium – preferred if sugammadex is available for reversal - Succinylcholine – preferred otherwise, if not contraindicated such as in denervating neuromuscular disease, malignant hyperthermia, hyperkalemia, recent burns or multi-trauma
6) Positioning	- Sniffing position in children >6 years if no c-spine concerns and shoulder roll in infants - Consider cricoid pressure in high-risk aspiration scenario is controversial - Backward, upward, rightward pressure (BURP) to aid in positioning the view of the glottis - Head-up positioning to improve functional residual capacity and preoxygenation in order to prolong time to desaturation
7) Placement	- Insert endotracheal tube and confirm position with end-tidal carbon dioxide and auscultation
8) Maintenance	- Set ventilator and provide ongoing sedation with opioids or benzodiazepines for example - Confirm depth of ETT with chest x-ray if in emergency department/pediatric ICU

POSITIONING


Infant
Small child
Older child/adult

ANATOMY


Pediatric
Adult
Tongue: Proportionally larger in children
Epiglottis: Longer and floppier in children
Vocal cords: Upward slant in children, forms the glottis which is the narrowest part of the adult airway
Cricoid: Narrowest part of pediatric airway
Trachea: Narrower and more flexible in children
Lungs: Reduced capacity in children

May 2024

Jillian Meyer (Medical Student, University of Alberta), Dr. Florian Rudolph (pediatric anesthesiologist, University of Alberta) for www.pedscases.com

RETINOPATHY OF PREMATURITY

Retinopathy of Prematurity (ROP): uncontrolled proliferation of retinal blood vessels in premature infants which may lead to blindness. Common in NICU settings!

PRESENTATION

- Red reflex in all neonates: look for **leukocoria** (white pupil)
- **Screen** for ROP in neonates with risk factors: **≤30 weeks GA**, birth weight <1500 g, systemic stressors (ex. hypoxemia, supplemental O_2)

SCREENING

Dilated retinal examination by an ophthalmologist

- If < 28 weeks GA: screen at 31 weeks corrected
- If ≥ 28 weeks GA: screen at 4 weeks of age
- Classify by zone (location) + severity

PATHOPHYSIOLOGY

Timeline of retinal maturation

- 16 wks GA: vessels emerge from optic disc
- 36 wks GA: vessels reach nasal edge of retina
- 9-10 months GA: vessels reach full 360°

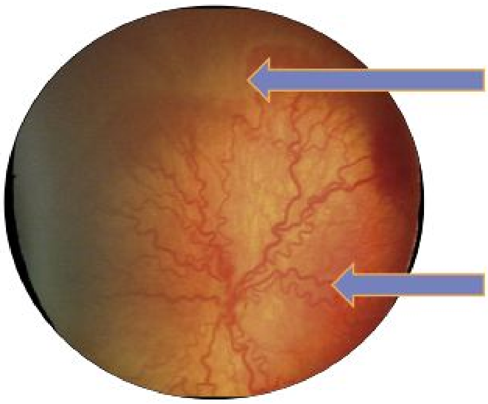
Pathophysiology of ROP

- Underdeveloped peripheral retina becomes ischemic → **VEGF** release → neovascularization → complications
- VEGF: vascular endothelial growth factor; promotes new blood vessel formation

COMPLICATIONS

- Bleeding of abnormal new vessels
- **Retinal detachment** → leukocoria
- Long-term: myopia, strabismus, cataract, glaucoma, amblyopia

This underscores the importance of checking for **red reflex** in all neonates! **Leukocoria** (white pupil) may indicate ROP (from retinal detachment complication), congenital cataract, retinoblastoma, or other causes. Consult ophthalmology!



Demarcation between avascular peripheral retina and vascular retina

Engorged and tortuous vessels with surrounding neovascularization

MANAGEMENT

If indicated to treat, sacrifice ischemic peripheral retina to **stop VEGF** production via:

- Pan-retinal photocoagulation (PRP) **laser**
- **Anti-VEGF therapy** (intraocular injection of monoclonal antibodies)

If present, treat retinal detachment (laser or surgery)

Follow-up, usually yearly, with ophthalmology throughout childhood after ROP diagnosis for long-term complications

July 2023
Kimberly Papp (Medical Student 2024, University of Alberta), Dr. Ian MacDonald (Pediatric Ophthalmologist, University of Alberta) for www.pedscases.com

RETT SYNDROME

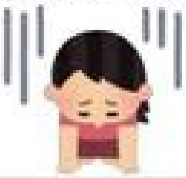
A **rare neurodevelopmental disorder** caused by a **mutation in the MECP2 gene**, resulting in insufficient or abnormal production of the MECP2 protein, which is **essential for normal brain function**. Primarily affecting **females**, it occurs in 1 in 8,500 births and follows an **X-linked dominant inheritance** pattern.

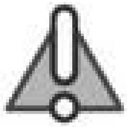
X – linked dominant

CLINICAL FEATURES

Growth:
- **Progressive microcephaly**
- Failure to thrive

Development:
- Initially, development is appropriate
- **Regression** occurs by 18 months of age





Neurologic:
- **Seizures**

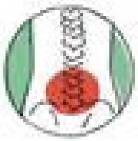
- Stereotypies
  - Clapping
  - **Hand-wringing**

- Gait ataxia
- Neuropsychiatric disturbance
  - Agitation
- **Sleep disturbance**

Gastrointestinal:
- Constipation

Extremities:
- Small hands and feet

Other:
- Prolonged QTc
- Bruxism (teeth grinding)
- Scoliosis



PROGRESSION THROUGH 4 STAGES

Early Onset	Rapid Regression	Plateau	Late Deterioration
6-18 months • Delayed development • Reduced interaction	1-4 years • Loss of speech, hand use, and mobility • Repetitive hand movements	2-10 years • Symptoms stabilize • Behavior improves • Motor issues persist	After 10 years old • Loss of mobility • Muscle weakness • Cognitive abilities often stable

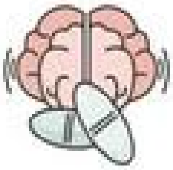
DIAGNOSIS

- Identification of **key clinical features**

Genetic Testing
- MECP2 molecular genetic testing

SURVEILLANCE

- **ECG** to rule out prolonged QTc
- **EEG** if suspected seizure activity

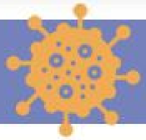


MANAGEMENT

Treatment focuses on **symptom control** with a **multidisciplinary approach**
i.e., antiepileptic drugs for seizures, melatonin for sleep disturbance, selective serotonin reuptake inhibitors (SSRIs) for agitation, fiber and PEG-3350 for constipation.

February 2025
Dr. Katharine V. Jensen (Pediatric Resident, University of Alberta) and Dr. Karen Forbes (Professor of Pediatrics, University of Alberta) for www.pedscases.com

ROUTINE CHILDHOOD IMMUNIZATIONS



Why is vaccination/immunization important?

Vaccines are crucial for ***preventing infectious diseases*** and their ***complications***, as well ***as potential outbreaks***. They protect not only the ***individual*** but also the community through ***herd immunity***, especially ***immunocompromised*** children who may not be able to receive vaccinations.

Potential Side Effects

- Common side effects – mild pain, redness, swelling at site, and mild fever.
- Rash (especially with MMR, MMRV, and varicella)
- Pain and anxiety
- Allergic reaction (very rare)
- *Consider delaying immunizations during moderate/severe illness*

AMINISTRATION TIPS & TRICKS

- Site selection:
    - Infants – anterolateral thigh
    - Older children – deltoid muscle of arm
- Positioning – Chest to chest, back to chest, lap sitting
- Strategies to minimize pain:
    - Being held by caregiver, breastfeeding, skin to skin, topical anesthetic, dextrose, distractions (eg. Bubbles, books, deep breathing)
- CARD (Comfort, Ask, Relax, Distract) System – https://immunize.ca/card-parents

CAREGIVER CONCERNS	EVIDENCE-BASED INFORTMATION
"Most diseases for which vaccines are given are not serious"	All of the diseases for which children are vaccinated are serious. They can cause serious illness, complications, and death. Many of these diseases also have no cure.
"My child doesn't need vaccines because no one gets these diseases anymore"	Thanks to vaccinations, rates of vaccine-preventable disease have declined in Canada. But with immunization rates declining, these disease may become more common, especially with travel to parts of the world where these disease rates are higher.
"If so many other people are vaccinated, my child does not need them"	If many parents choose not to vaccinate their children, the overall immunity drops, leading to the rapid spread of [illegible]ing on the immunity of others to protect the unvaccinated on[illegible]ne else is vaccinated. This is important to protect our immunocompromised patients.
"The MMR vaccine or Thimerosal in vaccines causes autism"	The MMR vaccine **does not** cause autism. There is no scientific evidence to suggest that any vaccine or vaccine preservatives (eg. Thimerosal) causes developmental disorders. Vaccines are safe and effective, and have gone through rigorous testing and investigation to prove their safety and effectiveness.

VACCINE COUNSELLING/ADDRESSING HESISTANCY TIPS (CALMS)

Communicate Clearly
Explain the benefits and safety of vaccines in simple terms.

Acknowledge Concerns
Listen to worries and respond with evidence-based information.

Lead by Example
Share personal or community stories about the positive impact of vaccines.

Maintain Trust
Build trust through honesty and transparency about vaccine information.

Support and Reassure
Offer continuous support and reassurance, providing regular reminders and follow up

Resources – https://caringforkids.cps.ca/handouts/immunization

November 2024

Lize-Mari Du Toit (Medical Student, University of Alberta), Dr. Karen Forbes (Pediatrician, University of Alberta) www.pedscases.com

SCABIES

What is scabies?

Scabies is a **contagious**, **pruritic** skin infection caused by reactions to the ***Scaroptes scabiei*** mite, its eggs or its feces. Scabies is not a reportable disease, so its true prevalence is difficult to ascertain.

PRESENTATION

- **Risk factors:**
  - Poverty
  - **Overcrowding**
  - Bed-sharing
  - Families with many children
  - Extremes of age (young, old)
  - Immunocompromised
  - Developmental delay
  - ***Lack of clean running water*** *is risk factor for secondary skin infections*

HISOTRY AND PHYSICAL EXAM

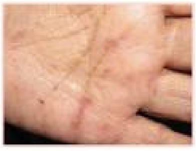
This is a clinical diagnosis

- **Pruritic** rash, worse at night
- **Burrows**, **erythematous papules** between the fingers, wrist flexures, elbows or armpits
- Does not usually occur above the neck
- **Symptoms in other household members**

Indigenous communities are disproportionately affected, resulting in significant morbidity

PATHOPHYSIOLOGY

- ☐ Direct skin-to-skin transmission of *Sarcoptes scabiei.* Limited transmission through fomites (ex: linen) since mite can only live 24-36 hours away from skin.
- ☐ Female adult mite burrows into epidermis and deposits eggs that hatch on day 2-4 and mature into adult mites over the next 10-14 days.
- ☐ Infected individuals develop a type I and IV hypersensitivity reaction to the mites, eggs, or excrement, illustrated by erythematous papules.



DIFFERENTIAL DIAGNOSIS

- Atopic dermatitis
- Insect bite
- Eczema
- Impetigo
- Tinea corporis (ringworm)
- Psoriasis

POTENTIAL INVESTIGATIONS

- Microscopic examination of skin scraping – scrape oil-coated blade across skin to obtain sample
- Burrow ink test – Lesion covered with ink, remove ink with alcohol. Ink remains tracking through burrows.
- Dermoscopy – Magnify and examine skin directly. *Difficult in remote setting*

POSSIBLE COMPLICATIONS:

Occur due to scratching

Secondary bacterial infection typically caused by ***S. aureus*** and ***S. pyogenes***

Risk of **sepsis** in **immunocompromised** individuals

MANAGEMENT

1st line: 5% permethrin cream.* Leave on for 12-14h, then bathe to remove. Repeat 1 week later. ***Use in patients >3 months***

Other topicals: 10% crotamiton lotion/cream, **sulphur precipitated in petroleum jelly** (safe in patients **<3 months**), benzyl benzoate

Other aspects of treatment:

- **All household members must be treated simultaneously to avoid re-infestation**
- **Bedding and clothing** should be **washed and dried** in a hot dryer **or sealed in a bag for >72 hours,** as mites typically cannot survive without human skin contact for longer than this

Itching may persist beyond treatment completion, and does not reflect a failure of treatment

November 2023
Emily Wener (Medical Student, University of Ottawa) & Dr. Alex MacKenzie MD, PhD (Pediatrician, Children's Hospital of Eastern Ontario) for www.pedscases.com

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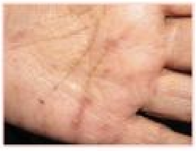

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November 2023
Emily Wener (Medical Student, University of Ottawa) & Dr. Alex MacKenzie MD, PhD (Pediatrician, Children's Hospital of Eastern Ontario) for www.pedscases.com

SCARLET FEVER

Bacterial infection caused by Group A streptococcal (GAS) with a characteristic diffuse, erythematous, "sandpaper-like" rash

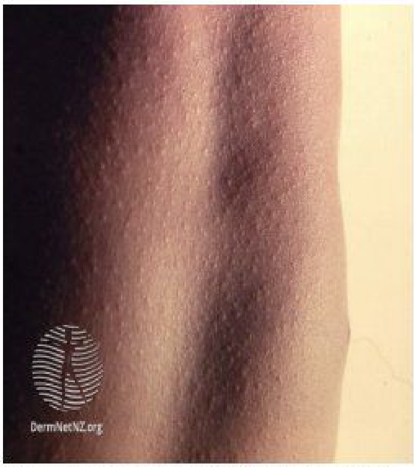
PATHOPHYSIOLOGY

- Group A beta-hemolytic streptococcus releases bacterial exotoxins, also known as erythrogenic toxins
- Leads to immune activation □ causes erythematous exanthem and enanthem
- Typically follows tonsillitis or pharyngitis but can occur with other sources of GAS infection such as burns and wounds.

DIAGNOSIS

Diagnosis is mainly **clinical**

- Confirm presence of GAS with rapid GAS testing +/- throat cultures
- May consider ASOT to confirm recent GAS infection

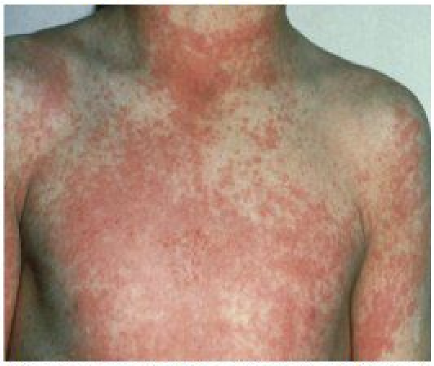


https://dermnetnz.org/topics/scarlet-fever

PRESENTATION

HISTORY

- Typically, school-aged children & adolescents (5-15 yo)
- Prodrome often includes sore throat, malaise, fevers/chills,
- 12-48 hours after fever usually marks the onset of a characteristic rash



https://www.nhs.uk/conditions/scarlet-fever/

PHYSICAL EXAM

Skin

- Blanchable erythema beginning on the neck, trunk, groin, axillae with subsequent diffuse involvement including the extremities
- Overlying small papules (sandpaper-like texture)
- Pastia's lines (petechial streaks caused by papules at pressure points in skin folds)
- Flushed cheeks with perioral pallor
- Desquamation in 7-10 days as rash resolves

Oropharyngeal/HEENT

- Tender cervical adenopathy
- Strawberry tongue (initial white coating, bright red papillae, edematous)
- Tonsillar erythema and exudate
- Palatal petechiae

DIFFERENTIAL DIAGNOSIS

- Other bacterial infections such as impetigo, erysipelas, and staphylococcal scalded skin syndrome (SSSS), toxic shock syndrome
- Kawasaki disease
- Viral exanthem (such as measles, chickenpox, hand foot mouth disease) all have which have specific clinical findings unique from scarlet fever
- Drug eruption

Potential complications of Group A Streptococcus infection include:

- **Suppurative:** peritonsillar abscess, otitis media, pneumonia & meningitis
- **Non-suppurative**: acute rheumatic fever, post-streptococcal reactive arthritis, acute post-streptococcal glomerulonephritis (PSGN)

MANAGEMENT

Antibiotics are the mainstay of treatment to help prevent suppurative complications and acute rheumatic fever

- **1st line:** penicillin or amoxicillin
- **Penicillin allergy**: 1st gen. cephalosporin or macrolide (ex: azithromycin)
- Duration of therapy: 10-14 days, 5 days for azithromycin
- Expect a response within 24-48 hours of starting antibiotics

July 2024
Jasmine Gill (Medical Student, University of Alberta) & Dr. Lillian Lim (University of Alberta)
for www.pedscases.com

SCHOOL REFUSAL

Definition: Refusal to attend school, distress with being at school. Often willing to do schoolwork at home.

Different from Truancy: Unexcused absences, dishonesty, lack of anxiety about absences, apathetic about meeting academic expectations

POTENTIAL CONSEQUENCES

- Underachievement
- Peer rejection
- Family tension
- Unemployment
- Unmanaged mental illness

Prognosis worsens with time if unmanaged

PRESENTATION

- Absenteeism, lateness, avoiding classroom
- Leaving school early to return home
- Emotional distress, behavioral outbursts, clinging
- Somatic symptoms (GI, autonomic, pain) **often resolve if the child is allowed to remain home**
- Common in early grades, after transition periods (new school, after school break, after illness)

CAUSAL FACTORS

- Depression
- Anxiety disorders
- Learning disorders, ADHD, language disorders
- Avoiding social or academic stressors (e.g. bullying)
- Chronic illness
- Poor sleep

FAMILY FACTORS

- Disruption, death, divorce, trauma, parent illness, child/sibling removal from home
- Parent: mental health, mistrust of school system, own negative school experience, devaluing education

MANAGEMENT

Must be a collaborative multidisciplinary effort with the goal of having the child return to school and activities **as soon as possible**

PHYSICIANS

- ☐ Address contributing factors
  - Medical conditions
  - Psychoeducational assessment
  - Sleep
  - Mental Health
- ☐ Psychoeducational support for parents and teachers
- ☐ If anxiety or depression, consider CBT or SSRIs
- ☐ If ADHD, consider stimulants
- ☐ Inpatient admission for intervention

CAREGIVERS

- ☐ Be empathetic but firm about return to school
- ☐ Track attendance goals
- ☐ Family therapy
- ☐ SPACE (Supportive Parenting for Anxious Childhood Emotions)
- ☐ Shift focus from child's distress to their efforts to attend school
- ☐ Address own anxiety
- ☐ Make staying home unrewarding

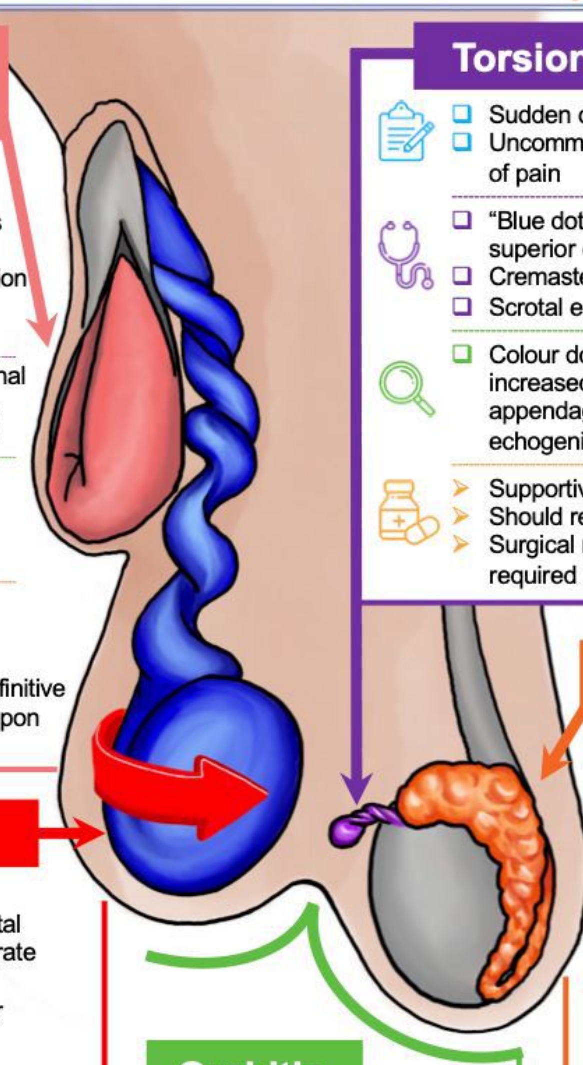
EDUCATORS

- ☐ Supportive classroom
- ☐ Address bullying
- ☐ Educational interventions and accommodations
- ☐ Identify adult and peer support figures
- ☐ Consistency with management plan developed with family and health care team

November 2024
Jasmin Majumdar (Medical Student, University of Alberta), Dr. Anisa Khaliq (Child and Adolescent Psychiatrist) for www.pedscases.com

SCROTAL PAIN

History · Physical Exam · Investigations · Management



Incarcerated Inguinal Hernia

- ☐ Infants: irritable, crying, poor feeding
- ☐ ?Previously reducible mass
- ☐ Vomiting, abdominal distension and/or constipation /obstipation if intestinal obstruction has occurred

- ☐ Firm, discrete, tender inguinal mass extending to scrotum
- ☐ Scrotal erythema & edema

- ☐ Labs and imaging of limited use
- ☐ Ultrasound occasionally helpful to confirm etiology

- Emergent reduction
- Most manually reducible, to OR if impossible in ER
- Once reduced, timing of definitive surgical repair depending upon age and degree of illness

Testicular Torsion

CANNOT MISS!

- ☐ Peak incidence: peri pubertal
- ☐ Pain: Sudden onset, moderate to severe, unilateral
- ☐ Occasional Hx of trauma or previous episodes of pain
- ☐ N/V common
- ☐ TWIST score

- ☐ High-riding testicle, horizontal lie
- ☐ Cremasteric reflex absent
- ☐ Scrotal erythema, edema, testicular tenderness
- ☐ May have reactive hydrocele

- ☐ Colour doppler ultrasound if low risk (low TWIST score) & Dx in question
  - Contraindicated if high likelihood and results in delay
- ☐ U/A generally not indicated and not needed for diagnosis

- High likelihood (high TWIST score) → immediate surgical exploration
  - Surgical detorsion and orchiopexy if viable
  - 97% salvage if **<6h** from sx onset
- Orchiopexy of contralateral testis

Torsion of Appendix Testis

- ☐ Sudden onset mild-severe pain
- ☐ Uncommon to have N/V or previous episodes of pain

- ☐ "Blue dot sign": palpable tender nodule at superior or inferior pole
- ☐ Cremasteric reflex present
- ☐ Scrotal erythema, edema, nontender testicle

- ☐ Colour doppler ultrasound: normal or increased blood flow to testis, torsed appendage shown as lesion of low echogenicity

- Supportive: analgesics, rest, scrotal support
- Should resolve in 5-10 days
- Surgical removal of appendix testis rarely required

Orchitis

- ☐ Occasionally bilateral testicular pain
- ☐ May show systemic manifestations of underlying infection

- ☐ Scrotal erythema, edema, tenderness, shininess of overlying skin

- ☐ Viral: mumps, rubella, coxsackie, and parovirus common
- ☐ Bacterial: brucellosis

- Supportive: rest, NSAIDs, ice packs

Epididymitis

- ☐ Bimodal incidence:
  - Childhood & sexually active
- ☐ Gradual onset pain
- ☐ Unlikely: Dysuria, frequency, discharge
- ☐ N/V more common with torsion
- ☐ No definitive discrimination with torsion

- ☐ Cremasteric reflex present
- ☐ Scrotal erythema, edema, epididymis tenderness

- ☐ Urinalysis: may be normal
- ☐ STI testing:
  - Urine NAAT for chlamydia and gonorrhea
  - Most common in pre-pubertal: coliforms and P. aeruginosa
- ☐ Colour doppler ultrasound: normal or ↑ blood flow

- No pyuria → no abx, supportive care
- Suspected STI cause: ceftriaxone IM x1 dose + doxycycline PO x10-14 days
- Suspected enteric cause: levofloxacin PO x10 days

Published May 2021
Sarah Park (Medical Student, University of Alberta), Dr. Peter Metcalfe (Pediatric Urologist, University of Alberta) and Dr. Mark Assmus (Urologist, Indiana University) for www.pedscases.com

SEBORRHEIC DERMATITIS

INTRODUCTION & PATHOGENESIS

- **Seborrheic** (seb-o-REE-ik) **dermatitis** is an inflammatory skin reaction that typically causes **mild**, **self-limited** dermatitis

The pathogenesis is a result of inflammation at the sebaceous glands and is thought to be multifactorial including:

- Reaction to *Malassezia spp.*, a yeast that lives on skin surfaces
- Sebum production and hormonal influences
- Environmental (eg. cold or dry weather, exposure to irritants)

DIAGNOSIS

Clinical Diagnosis

- ☐ **S**ize – Variable (rash)
- ☐ **C**olor - erythematous, white and yellow scale
- ☐ **A**rrangement – see 'Clinical Presentation'
- ☐ **L**esion Morphology - dermatitis
- ☐ **D**istribution – scalp and skin folds
- ☐ May overlap with atopic dermatitis leading to head and neck dermatitis. Treatment principles are similar.

DIFFERENTIAL DIAGNOSIS

- **Atopic Dermatitis**
- **Psoriasis**
- **Contact dermatitis**
- **Langerhans cell histiocytosis**

CLINICAL PRESENTATION

Primarily occurs in a bimodal distribution:

- Infantile (2 weeks -12 months)
  - Occurs **in 70% of infants <3 months**
  - Usually, self-limiting
- **Adolescence**
  - Consider screening for immunodeficiency if presenting in adolescence
  - **May proceed into adulthood**

Appearance

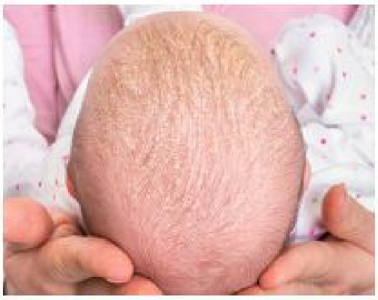
- **Scaly, greasy and patchy** dermatitis
- Erythematous skin with white and yellow flakes
- Inflammation/occasional swelling
- Skin flakes (dandruff) may persist in adulthood
- **Skin of colour may present hypopigmented**

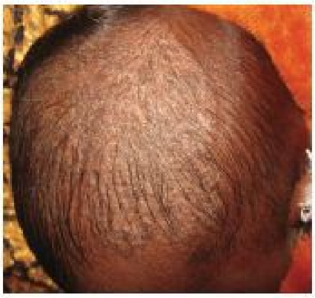
Distribution Pattern

- Often presents on the **scalp** in a cap distribution (aka "Cradle Cap")
- Can present in other areas such as face, trunk, buttocks, ears, groin and axilla

Symptoms

- Child usually presents unbothered
- May be pruritic/symptomatic if more severe





MANAGEMENT

1. Can be left **untreated** if child is unbothered (infantile) – often self resolves
2. Home treatment: use of **fragrance-free gentle soap wash and moisturizer,** (+/- over the counter dandruff or ketoconazole shampoo). Using Olive oil/ oil can cause proliferation of yeast and worsen the condition
3. Topical **antifungals** (e.g., ketoconazole), low dose topical **corticosteroids**
   - if resistant/severe, consider dermatologist referral

Goals: reduce itch, remove scales, decrease inflammation

May 2022

McKenzie Van Eaton (Medical Student, University of Saskatchewan), Rachel Asiniwasis (MHSc, MD, FRCPC)
for www.pedscases.com

SEVER'S DISEASE

Sever's disease, also known as calcaneal apophysitis, is **painful inflammation** of the **calcaneal apophysis**. It is the most common cause of heel pain in growing children.

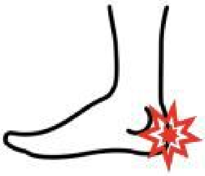
PRESENTATION

- Pain with **heel compression**
  - **Worsens with weight bearing, running, or jumping**
  - Pain **resolves with rest**
  - **No pain** in the **morning**
- No swelling, erythema, or skin changes
- Tight Achilles tendon
- May have limp

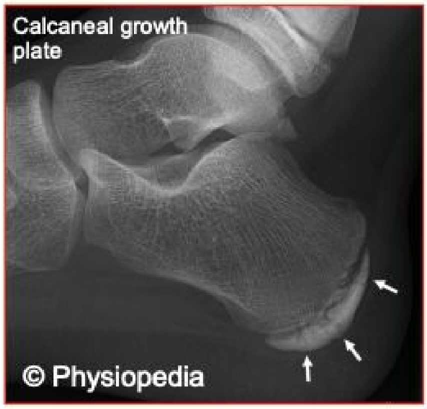
Can be unilateral or bilateral

PHYSICAL EXAM

- **SEADS**: inspection for **s**welling, **e**rythema, **a**trophy, **d**eformity, and **s**cars
- Tenderness on palpation of heel
- Worse heel pain standing tiptoe
- Decreased dorsiflexion
- Biomechanical abnormalities
- Gait assessment; assess for limp



Squeeze test: mediolateral compression of the calcaneal growth plate elicits pain



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PATHOPHYSIOLOGY

- Affects **active children,** especially during **pubertal growth spurt** or at the start of the sports season
- Caused by **repetitive microtrauma** on the **unossified apophysis**
- Due to **traction of the Achilles tendon at the apophysis**

RISK FACTORS

- High physical activity
- Running on hard surfaces
- Poor footwear
- Genu varum, pes cavus, or pes planus
- Weak dorsiflexion
- Achilles tightness
- Obesity

DIAGNOSIS

Sever's disease is a CLINICAL DIAGNOSIS

Generally, investigations are unnecessary; however, in situations where a diagnosis is not certain, the following imaging studies can be considered:

- **X-ray**: exclude other trauma
- **Ultrasonography:** can reveal fragmentation of the calcaneal growth plate. Can exclude Achilles tendinopathy and bursitis.
- **MRI**: can localize inflammation to the calcaneal apophysis

MANAGEMENT

- **Stop** or **modify activities** that cause pain
- **RICE**: **r**est, **i**ce, **c**ompression, and **e**levation
- **Taping** the arch and heel
- Nonsteroidal anti-inflammatory drugs (**NSAIDs**) PRN
- Silicone heel cup
- **Shoes** with good medial arch support or arch support cushions
- Over the counter **orthotics** to correct secondary foot malalignment

August 2024
Dr. Katharine V. Jensen (Pediatrics Resident, University of Alberta) and Dr. Jeanine McColl (Pediatric Rheumatologist, University of Calgary) for www.pedscases.com

SICKLE CELL DISEASE: ACUTE COMPLICATIONS

Complications of sickle cell disease arise because Hemoglobin S polymerizes and becomes rigid upon deoxygenation, causing vaso-occlusion and hemolysis.

Triggers for hypoxic states include:

- **infection**
- **dehydration**
- **stress**
- **extreme temps**

Vaso-Occlusive Crisis (Acute Pain Episode)

Vaso-occlusion → hypoxic-ischemic and reperfusion injury

Presentation: Acute pain (commonly long bones, chest, abdomen).

Investigations: Usually a clinical diagnosis, but rule out other causes.

Initial Management:

- Analgesia: acetaminophen, NSAIDs, opioids
- IV fluids

Stroke

Ischemic stroke most common in children, hemorrhagic in adults. High risk of stroke recurrence (60-90%) without secondary prevention.

Presentation: Headache, nausea & vomiting, focal neurologic deficits, seizures, altered level of consciousness.

Investigations: CT/MRI

Initial Management:

- Stabilize vitals
- Red blood cell exchange transfusion

Sepsis

Primary cause of mortality in children. ↑ risk of sepsis from encapsulated organisms as functionally asplenic.

Presentation: Fever and unwell-appearing. Highest risk if <5 years old.

Investigations: CBC+diff, blood culture, other infectious workup as appropriate.

Initial Management:

- 3rd gen cephalosporin +/- vancomycin

Splenic Sequestration

Vaso-occlusion leads to trapping of erythrocytes with rapid painful enlargement of the spleen and acute drop in hemoglobin.

Presentation: Hypotension, tachycardia, LUQ pain, splenomegaly.

Investigations: CBC+diff with retic count

Initial Management:

- Transfusion of pRBCs in small aliquots (beware auto-transfusion from spleen).
- Reassess vitals, hemoglobin, and spleen size regularly.

**If recurrent or life-threatening, consider splenectomy after acute event has resolved.*

Acute Chest Syndrome

Clinically defined as new infiltrate on CXR with respiratory symptoms or fever. Often precipitated by infection.

⚠ May progress rapidly to respiratory failure

Presentation: Chest pain, cough, SOB, tachypnea, fever, hypoxia.

Investigations: CXR, CBC+diff with retic count

Initial Management:

- Optimize ventilation. Incentive spirometry.
- Hydration and analgesia.
- Antibiotics: 3rd generation cephalosporin and macrolide (cover *Mycoplasma, S. pneumoniae*).
- Simple or exchange red blood cell transfusion.

Published December 2020
(Genie) Eugene Kwon (Medical Student, University of Alberta) and Dr. Catherine Corriveau-Bourque (Pediatric Hematologist, University of Alberta) for www.pedscases.com

SICKLE CELL DISEASE: OVERVIEW

Definition

Sickle cell disease (SCD) is a group of inherited red blood cell disorders caused by abnormal hemoglobin resulting in acute and chronic complications.

Pathophysiology

- Point mutation in β-globin gene changing amino acid Glu → Val at position 6, resulting in HbS
- HbS polymerizes → red blood cell sickling → vaso-occlusion, changes in blood viscosity, hemolysis, endothelial dysfunction, and inflammation

Presentation

Acute Complications

- Vaso-occlusive Crisis (Acute Pain Episode)
- Acute Chest Syndrome
- Sepsis
- Splenic Sequestration
- Stroke
- Dactylitis
- Priapism

Chronic Complications

- Poor growth
- Renal disease
- Retinopathy
- Osteonecrosis
- Functional asplenia
- Cholelithiases
- Learning & cognitive difficulties
- Silent cerebral infarcts
- Chronic lung disease
- Pulmonary hypertension
- Congestive heart failure

Diagnosis

Newborn screening in many provinces
Hemoglobinopathy investigation

Management

Targeted towards managing & preventing complications

⚠ Involve hematology prior to any blood product transfusion or procedure ⚠

Education

Teach families splenic palpation and to urgently seek care with rapid splenic enlargement/fever

Prophylactic Penicillin

Prophylaxis against encapsulated organisms as functionally asplenic. Continue to minimum age 5 years

Immunizations

Routine immunizations + PPSV 23

Transcranial Doppler

Screening for stroke prevention begins at age 2

Hydroxyurea

Increases total fetal Hb, decreases WBC. Decreases admissions for Acute Chest Syndrome and pain episodes.

Red Blood Cell Transfusions

Simple or exchange transfusions. Indicated only in certain clinical situations as high risk alloimmunization. Reduces the HbS%.

Hematopoietic stem cell transplant

Currently, the only cure for SCD

Published December 2020
(Genie) Eugene Kwon (Medical Student, University of Alberta) and Dr. Catherine Corriveau-Bourque (Pediatric Hematologist, University of Alberta) for www.pedscases.com

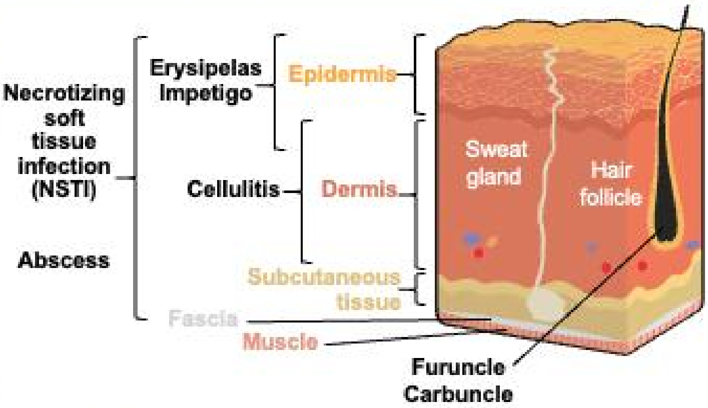
SKIN AND SOFT TISSUE INFECTIONS

SIGNS & SYMPTOMS

- Erythema, warmth, tenderness, pain, fever, purulence
- **Systemic signs:** Temp. >38°C, HR >90, RR >24 or WBC <12,000 or <300 cells/uL

HISTORY

- Onset of signs/symptoms? Progression?
- Association with trauma?
- Burn(s), frostbite, pressure ulcer, post-surgical?
- Environmental risks? Vaccination history?
- Severity of pain? Radiation?
- Loss of function? Joint involvement?

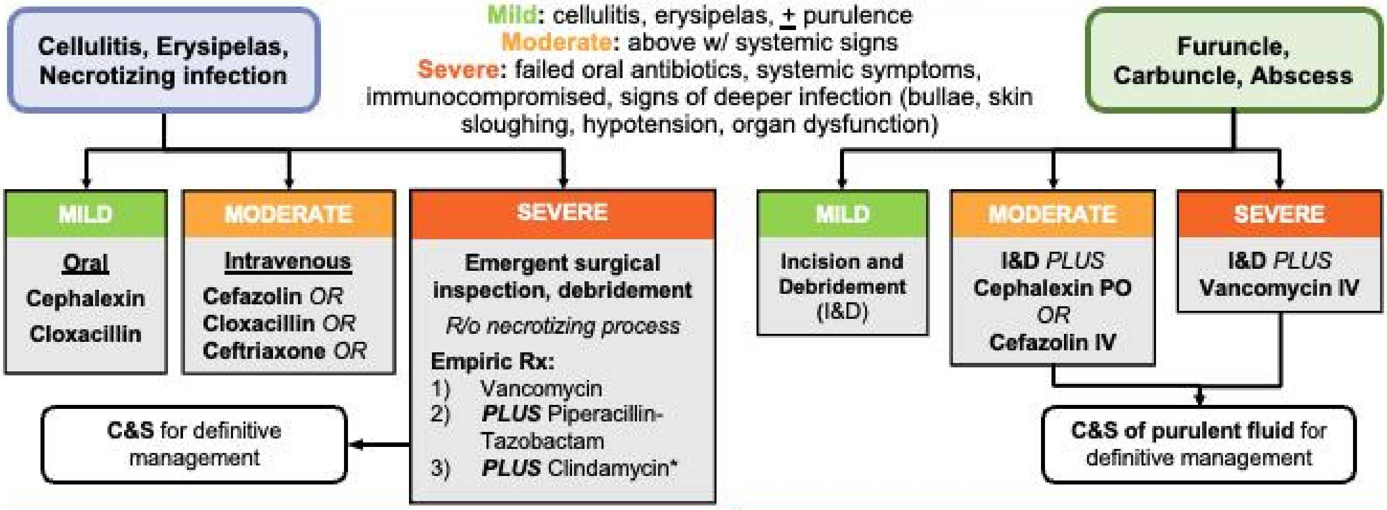


Non-Purulent SSTI

Erysipelas	Associated with fever, well demarcated erythema	*Group A Streptococcus* (GAS)	
Impetigo	**Non-bullous:** painless, erythematous base w/ honey-crusted exudate on face/limbs	*S. aureus*, GAS	
	Bullous: clusters of bullae/solidary lesions of exudate ± desquamation	Toxin producing *S. aureus*	
Cellulitis	Edema, pain, poorly demarcated erythema. Orbital cellulitis is a medical emergency!	GAS, *S. aureus*	
Necrotizing Soft Tissue Infection (NSTI) ⚠	Generally, rapidly evolving, pain out of proportion, erythematous rash w/ fever, toxic appearance, & thrombocytopenia. Hemodynamically unstable.	**Monomicrobial** *S. aureus* most common	**Polymicrobial** GAS, MRSA, VRE, *Clostridium*

Purulent SSTI (Drainable Collection)

Cutaneous / Deep Soft Tissue Abscess	Collection of pus	*S. aureus* (community-acquired MRSA), GAS
Furuncles, Carbuncles ± Cellulitis	**Furuncles (boils)** are skin abscesses that involve a hair follicle. **Carbuncles** are clusters of furuncles.	*S. aureus* (including MRSA), GAS



Mild: cellulitis, erysipelas, ± purulence
Moderate: above w/ systemic signs
Severe: failed oral antibiotics, systemic symptoms, immunocompromised, signs of deeper infection (bullae, skin sloughing, hypotension, organ dysfunction)

Cellulitis, Erysipelas, Necrotizing infection

- **MILD**: **Oral** **Cephalexin** **Cloxacillin**
- **MODERATE**: **Intravenous** **Cefazolin** *OR* **Cloxacillin** *OR* **Ceftriaxone** *OR*
- **SEVERE**: **Emergent surgical inspection, debridement**
  R/o necrotizing process
  Empiric Rx:
  1) Vancomycin
  2) ***PLUS*** Piperacillin-Tazobactam
  3) ***PLUS*** Clindamycin*
  → **C&S** for definitive management

Furuncle, Carbuncle, Abscess

- **MILD**: **Incision and Debridement** (I&D)
- **MODERATE**: **I&D** *PLUS* **Cephalexin PO** *OR* **Cefazolin IV**
- **SEVERE**: **I&D** *PLUS* **Vancomycin IV**

→ **C&S of purulent fluid** for definitive management

NSTIs IN PEDIATRICS

⚠

1) No skin manifestations in up to 50% of cases.
2) A prolonged prodrome or perceived slow onset should NOT rule out an NSTI.
3) May present with multifocal sites. Always check for additional sites during the physical exam.

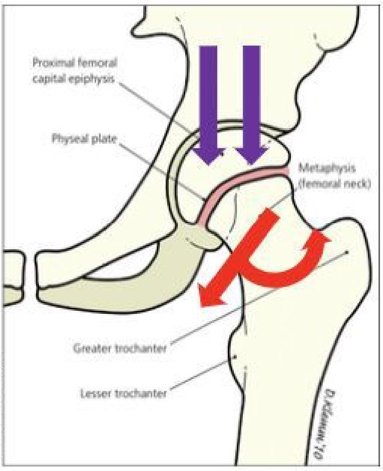
EAGLE EFFECT

The **Eagle Effect** is the paradoxical effect of reduced penicillin efficacy at higher antibiotic doses. **Clindamycin** also inhibits bacterial toxin production.

Published December 2020
Alexander Perry (Medical Student 2021, University of Alberta), Courtney Wilkes (Plastics PGY-5, University of Alberta) & Dr. Jaret Olson (Pediatric Plastic Surgeon, University of Alberta) for www.pedscases.com

SLIPPED CAPITAL FEMORAL EPIPHYSIS

PATHOPHYSIOLOGY


Proximal femoral capital epiphysis
Physeal plate
Metaphysis (femoral neck)
Greater trochanter
Lesser trochanter
D.Klemm '10
ILLUSTRATION BY DAVID KLEMM

Increased load on the proximal femoral physis causes **anterior** translation and **external** rotation of the metaphysis in an at-risk patient

PRESENTATION

Presentation

- Painful limp
- Pain may localize to the groin, thigh, ipsilateral knee or hip (posterior or lateral side)

Epidemiology

- Children ages 8-16
- M:F; 2:1.4

Risk Factors

- Obesity, endocrine disorders
- Adolescent growth spurt

PHYSICAL EXAM

MSK Exam

- Limited active internal rotation on affected side
- Passive internal rotation may elicit pain
- Loss of hip flexion, abduction, and internal rotation
- Antalgic gait

Drehmann Sign

- With passive flexion of the hip to 90 degrees, external rotation occurs

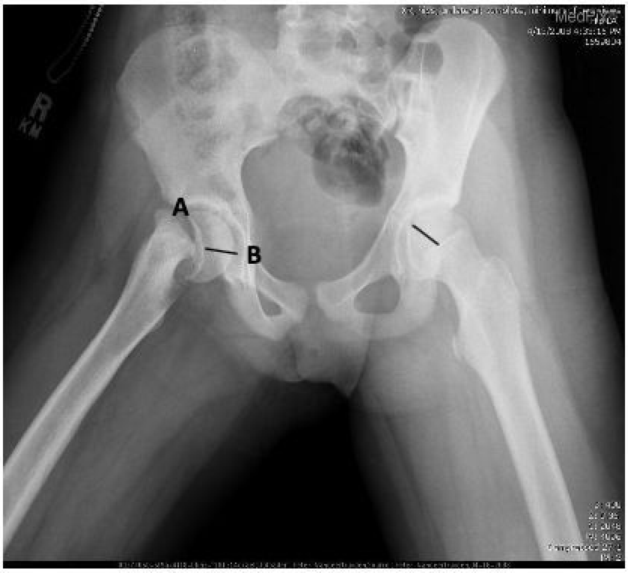
DIAGNOSIS & RADIOGRAPHY

Radiography (Lateral View)

☐ Widening of the physis (**A**)
☐ Decreased epiphyseal height compared to contralateral side (**B**)
☐ Loss of anterior concavity of the head-neck junction
☐ Note the medial and posterior displacement of the physis relative to the metaphysis

Diagnosis

☐ Suspect SCFE Dx with characteristic painful limp
☐ Diagnosis is made with plain film x-rays (AP and lateral 'frog leg' views)



MANAGEMENT

- Ensure patient uses a wheelchair or weight bearing crutches prior to orthopedic evaluation
- Definitive treatment: Surgical fixation
- Goal of surgery: Stabilize epiphysis and promote early closure of the growth plate via percutaneous screw fixation

COMPLICATIONS

- Long-term degenerative osteoarthritis
- Avascular necrosis of the femoral head
- Chondrolysis
- Limb length discrepancy

July, 2021

Timber Gillis (Medical Student 2024, University of Alberta), Dr. Michelle Noga (Professor of Radiology & Diagnostic Imaging, University of Alberta) for www.pedscases.com

SMOKING PREVENTION AND CESSATION

In North America, tobacco use is the **number one cause of preventable disease and mortality**. Nearly 90% of smokers who are adults first smoked a cigarette before turning 18.

Although smoking rates have declined, E-cigarettes are now impacting how teenagers interact with tobacco products; youth are more vulnerable and are at higher risk for smoking addiction compared to adults.

Forms of tobacco

Smoke: Cigarette, cigar, hookah, bidi, kretek

Smokeless: Chewing tobacco, dissolvable tobacco, e-cigarettes, snuff

FACTORS CONTRIBUTING TO SMOKING INITIATION

- Easy access to tobacco
- Many forms of tobacco
- Misinformation about the health consequences of smoking
- Low socio-economic status
- Sexual minority or Indigenous background
- Peer and family influence, including lack of parental support
- Substance abuse
- Adverse childhood experiences

CONSEQUENCES OF SMOKING

- Craving, tolerance, and withdrawal leading to dependence
- Negative effects on brain and development
- Reduced executive cognitive function in adulthood
- Increased risks for future substance use
- Increased impulsivity, decreased attention
- Bad smell, poor breath, early wrinkles
- Increased risk of developing chronic illnesses such as COPD, cardiovascular disease and adverse health effects on youth with pre-existing chronic illnesses
- Major risk factor for developing lung cancer

Motivational interviewing 5A's for smoking cessation

Ask Advise Assess Assist Arrange

PREVENTION INTERVENTIONS

- **Age-appropriate** information and **counselling** about tobacco use
- Encourage **family involvement**
- **5A's** method for motivational interviewing
- Support **tax raises** and **bans** on tobacco
- Encourage **government** and **school-based interventions**

CESSATION INTERVENTIONS

- Individual or group counselling and **motivational interviewing**
- **Cognitive behavioural therapy**
- Mobile phone interventions
- Self-help materials
- **Nicotine-replacement therapy**
- Pharmacotherapy: Bupropion, Varenicline

May 2024
Vaishvi Patel (Medical Student), Ronan Noble (Medical Student), Dr. Jackie Lee (Pediatrician, University of Alberta) for www.pedscases.com

SPEECH & LANGUAGE DELAY

PREVALENCE

- 3-10% of children
- 3-4x more common in boys than girls

RISK FACTORS

- Primary hearing deficit
- Secondary hearing deficit (e.g., recurrent acute otitis media, otitis media with effusion)
- Craniofacial abnormality
- Prematurity
- Low birthweight
- Family history

- Multi-lingual environment is not associated with speech/language delay

ASSOCIATED CONDITIONS

☐ Autism spectrum disorder
☐ Cerebral palsy
☐ Hearing loss
☐ Intellectual disability

MANAGEMENT

☐ Refer to speech language pathologist & audiologist ASAP
☐ Caregiver support for child's language development
    ☐ Maintain eye contact with child
    ☐ Encourage child to focus on caregivers articulating different sounds
    ☐ Encourage child to vocalize their request, as opposed to anticipating their need and offering proactively

IMPORTANT

- If delay is not properly addressed,
  - Higher risk for learning disability
  - Difficulty in reading and writing
  - Difficulty in school
- Timely intervention is key

DIAGNOSIS

Failure to meet age-appropriate milestones

SPEECH	LANGUAGE
▪ Delay in development of sound production **Example** ▪ Stuttering ▪ Articulation challenges	▪ Delay in development or use of language **Example** ▪ Expressive language delay ▪ Receptive language delay ▪ Difficulty with grammar or vocabulary

Milestones

Age	Speech / Language
6 mo	Responds to voice Babbles
1 yo	Follow **1** step commands Says **1** word (in addition to "mama", "dada")
2 yo	Follow **2** step commands 50+ words Says **2**-word combinations Speech ½ intelligible*
3 yo	Follow **3** step commands 200 words Says **3**-word combinations Speech ¾ intelligible*
4 yo	Follow **4** step commands Says **4**-word combinations (full sentences) *intelligible to strangers Speech 100% intelligible*

RED FLAGS

☐ >6 mo: limited/no vocalization
☐ 12 mo: not turning to names or words
☐ 15 mo: no single words

February 2024
Selina Chow (Medical Student, University of Toronto), Dr Rhea D'Costa (Developmental Pediatrician, University of Toronto) for www.pedscases.com

STATUS EPILEPTICUS

Status epilepticus (SE) is a **medical emergency** that is associated with significant morbidity and mortality.

TYPES OF STATUS EPILEPTICUS

CONVULSIVE STATUS	NON-CONVULSIVE STATUS	FOCAL STATUS	ABSENCE STATUS
≥ 5 minutes of **continuous** convulsive seizure OR **≥ 2** discrete seizures **without return to baseline** OR repeated seizures for **≥ 30 minutes.**	**≥ 10 minutes** of continuous seizure OR **≥ 30 total minutes of ictal EEG activity** in any given hour. These patients are at risk for convulsive status.	Focal epileptic seizure that lasts **≥ 30 minutes** OR **repeated focal epileptic seizures** (≥ 30 minutes) with **incomplete recovery** between seizures.	Prolonged, generalized **absence** seizure that usually last for **hours** to **days.** Cardinal symptom is **altered level of consciousness.**

Convulsive status is the most common type of status epilepticus (SE). > 50% of SE episodes occur in children with **no prior seizure history.**

COMMON ETIOLOGIES OF STATUS EPILEPTICUS

ACUTE SYMPTOMATIC (17-52%)		REMOTE (16-39%)		OTHER
➢ CNS infection ➢ Metabolic (hypoglycemia, hyperglycemia, hyponatremia, hypocalcemia) ➢ Stroke	➢ Hemorrhage ➢ Non-compliance with AEDs ➢ Overdose ➢ Toxins	➢ Progressive neurodegenerative disorders ➢ Cerebral migrational disorders	➢ Perinatal hypoxic-ischemic encephalopathy ➢ Cerebral dysgenesis	➢ **Epilepsy** ➢ **Idiopathic** (5-19%) ➢ **Prolonged febrile convulsions** (23-30%) ➢ Trauma

HISTORY

- **Seizure history:** pre-ictal, ictal, and post-ictal phases. Ask about duration, focal symptoms, provoking events, and use of anti-convulsant medications.
- **Past medical history:** previous seizures or history of epilepsy or other neurological disorders.
- **Family history**: seizures or epilepsy.
- **Illness symptoms:** fever, nausea, vomiting, diarrhea, rash.
- **Trauma, accidental** and **non-accidental injury**
- Medications (**AED**)
- **Toxins**

PHYSICAL EXAM

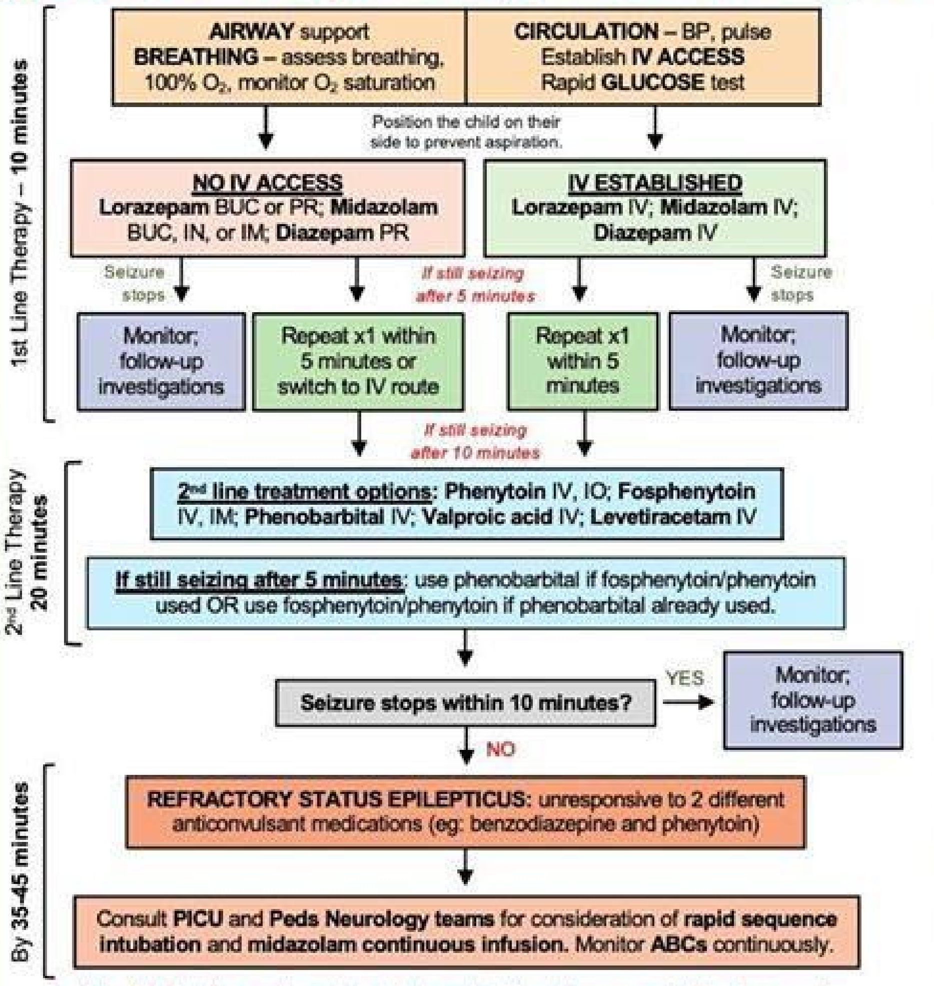
- **ABCs, vitals, level of consciousness, GCS**
- Rule out **CNS infections**
- **General physical exam:** source of infection (eg: otitis media, upper respiratory tract, lungs, GI tract, urinary tract, or sepsis).
- **Neurological exam**
- Indications of **toxidrome**

INVESTIGATIONS

- Glucose
- CBC with differential
- Electrolytes
- Ca^{2+}, Mg^{2+}, P
- Liver function tests
- Toxicology screen
- Anticonvulsant level
- Electroencephalogram (EEG)
- Head CT or MRI
- Urine, blood, CSF cultures

MANAGEMENT OF STATUS EPILEPTICUS IN HOSPITAL

GOAL: **stop the seizure** and **prevent brain injury**. Then determine the underlying cause.



1st Line Therapy – 10 minutes

AIRWAY support
BREATHING – assess breathing, 100% O_2, monitor O_2 saturation

CIRCULATION – BP, pulse
Establish **IV ACCESS**
Rapid **GLUCOSE** test

Position the child on their side to prevent aspiration.

NO IV ACCESS
Lorazepam BUC or PR; **Midazolam** BUC, IN, or IM; **Diazepam** PR

IV ESTABLISHED
Lorazepam IV; **Midazolam** IV; **Diazepam** IV

Seizure stops → Monitor; follow-up investigations

If still seizing after 5 minutes → Repeat x1 within 5 minutes or switch to IV route (no IV access); Repeat x1 within 5 minutes (IV established)

Seizure stops → Monitor; follow-up investigations

If still seizing after 10 minutes

2nd Line Therapy 20 minutes

2nd line treatment options: **Phenytoin** IV, IO; **Fosphenytoin** IV, IM; **Phenobarbital** IV; **Valproic acid** IV; **Levetiracetam** IV

If still seizing after 5 minutes: use phenobarbital if fosphenytoin/phenytoin used OR use fosphenytoin/phenytoin if phenobarbital already used.

Seizure stops within 10 minutes?

YES → Monitor; follow-up investigations

NO

By 35-45 minutes

REFRACTORY STATUS EPILEPTICUS: unresponsive to 2 different anticonvulsant medications (eg: benzodiazepine and phenytoin)

Consult **PICU** and **Peds Neurology teams** for consideration of **rapid sequence intubation** and **midazolam continuous infusion.** Monitor **ABCs** continuously.

Note: this algorithm applies only to infants and children. Management differs for neonates.

May 2020
Katharine V. Jensen (Medical Student 2021, University of Alberta) &
Dr. Natarie Liu (Neurologist & Epileptologist, University of Alberta) for www.pedscases.com

STOMATITIS

Stomatitis: inflammation of the oral mucosa (including inner cheeks, gums, lips, and tongue) that is characterized by the presence of painful ulcerations or lesions inside the mouth

DIFFERENTIAL DIAGNOSIS

Recurrent Aphthous Stomatitis	- Benign condition that may regularly reoccur - Sole physical finding: ≥1 painful oral ulcer
Infection	**Viral** - Most common infectious cause - Common Agents: · HSV1 · Coxsackievirus causing HFMD or herpangina **Bacterial** - Uncommon if otherwise healthy **Fungal** - Common Agents: · Candida
Contact Stomatitis	**Irritants** - Ex. excessive hot/cold exposure, acidic or basic substances, smokeless tobacco **Allergens** - Ex. preservatives, oral flavourings
Systemic Disorders	- Behçet's disease - IBD - Celiac disease - Diabetes mellitus - Systemic lupus erythematosus - Dermatomyositis - PFAPA - Cyclic neutropenia - Nutritional deficiencies
Iatrogenic	- Chemotherapy or radiation therapy

REFER TO SPECIALIST WHEN:

- Underlying cause is uncertain
- Oral lesions persist >2 weeks
- Other signs of systemic disease

PRESENTATION

Clinical Features

Features vary per etiology

- Red or white oral lesion(s) often with erythematous halos
- Erosions or ulcerations
- Fissuring at corners of mouth
- Mouth pain or burning

Signs of Systemic Disease

- Fever
- Failure to thrive
- Lymphadenopathy
- Organomegaly
- Dermatologic or joint abnormalities

INVESTIGATIONS

- **Most cases:** lab evaluations are not helpful in diagnosis or management
- **If persistent lesions with other systemic findings**: CBC, ESR, serum iron/folate/vitamin B12/zinc

MANAGEMENT

TREATMENT GOALS:

Maximize pain relief
Prevent dehydration

Agents for Pain Relief

- Systemic ibuprofen or acetaminophen
- Topical Substances
  - Saline rinse
  - Diphenhydramine-aluminum hydroxide with magnesium hydroxide-viscous lidocaine compounded in various combinations ("*Magic Mouthwash*")
  - Benzocaine
  - Pectin
  - Kaolin

Antimicrobials

** Antimicrobials are generally not indicated*

** Oral acyclovir is not indicated in most cases of stomatitis; it may be indicated in children presenting early in disease course with severe HSV stomatitis or in immunocompromised patients*

** Topical acyclovir is ineffective in an otherwise healthy child with herpes stomatitis*

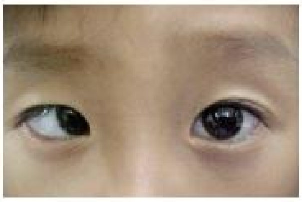
July 2024
Emily Cheung (Medical Student, McMaster University), Dr. Erin Boschee MD FRCPC (Pediatrician, Stollery Children's Hospital) for www.pedscases.com

CHILDHOOD STRABISMUS

Up to 5% of all children may have some type or degree of strabismus. Strabismus is defined as an abnormal alignment of the eyes. If onset is in childhood, strabismus is often comitant - the misalignment or deviation of the eyes is the same in all gaze positions. Because untreated strabismus is a cause of amblyopia* and irreversible blindness, suspected strabismus should be promptly referred to ophthalmology.

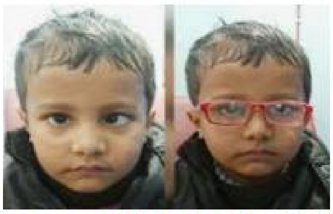
COMMON CHILDHOOD STRABISMUS TYPES AND PRESENTATIONS

1. Congenital esotropia: The constant inward turning of one or both of the eyes. This misalignment of the eyes is often identified prior to 6mo of age.



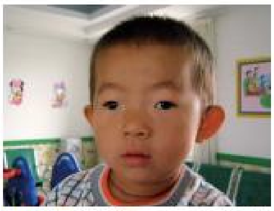
Esotropia © Community Eye Health, CC BY-NC 2.0

2. Accommodative esotropia: The inward turning of one or both of the eyes when trying to focus on close objects. Onset is typically between 6mo-7yrs of age and starts as an intermittent strabismus that becomes constant with time.



Accommodative Esotropia © IAPB/VISION, CC BY-NC-SA 2.0

3. Congenital exotropia: The constant outward turning of one or both of the eyes. Typical presentation is before 6mo of age and is associated with craniofacial malformations and neurologic abnormalities.



Exotropia © Community Eye Health, CC BY-NC 2.0

PHYSICAL EXAM: TESTS THAT HIGHLIGHT STRABISMUS

- Ophthalmoscopy and Red Reflex (the misaligned eye may have a brighter and lighter red reflex)
- Corneal Light Reflex (light may not be centered on the pupil of the deviated eye)
- Cover Test (if the uncovered eye moves to pick up fixation, this suggests a strabismus)
- Extraocular eye movement testing (misalignment of the eye may be observed in all gaze positions)

Cover Test © 176th Wing, Alaska Air National Guard, CC BY 2.0

***Amblyopia** is a decrease in visual acuity due to a disruption in how the brain and eyes communicate. Strabismus is the most common cause of amblyopia.This disruption can cause a progressive decrease in visual acuity with varying degrees of correctability.

NEXT STEPS

If strabismus is suspected, ophthalmology needs to be consulted.

November 2023
Megan Pohl (Medical Student, University of Alberta), Christopher Rudnisky (MD MPH FRCSC, Department of Ophthalmology & Visual Sciences, University of Alberta)

STREPTOCOCCAL PHARYNGITIS

Key Points

- Common clinical features of acute Strep pharyngitis:

Cervical lymphadenopathy | **Pharyngeal exudate** | **Fever (>38°C)** | **No cough/ rhinorrhea** | **Palatal petechiae**

3-14 year-olds most susceptible | less common in younger & older

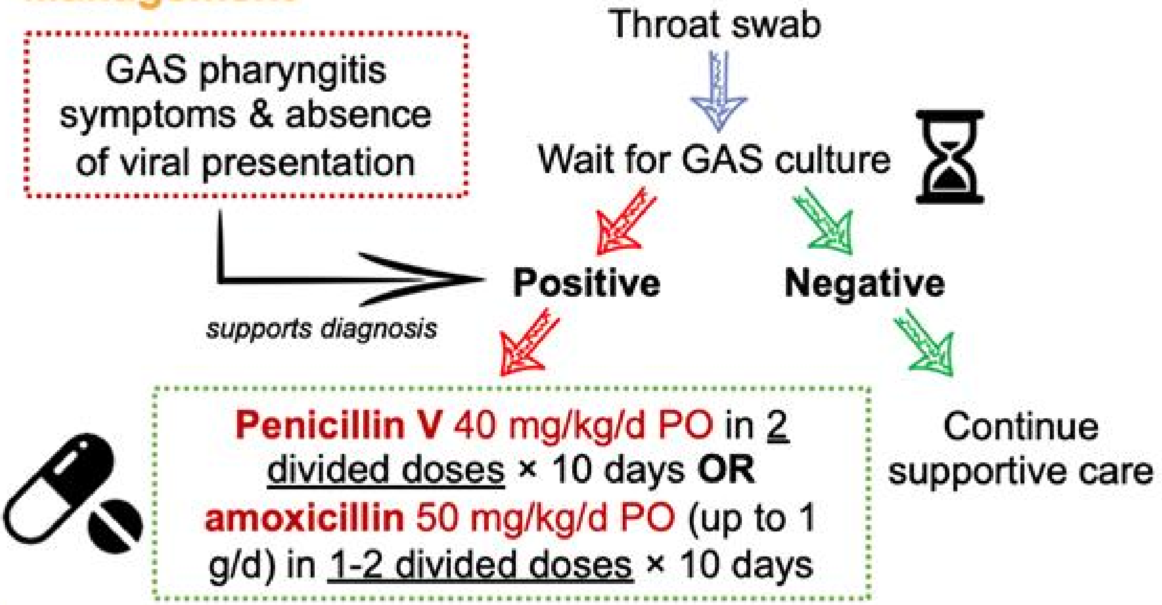
- Avoid unnecessary testing if clinical picture suggests classic viral etiology (eg. presence of cough, rhinorrhea, or hoarseness)
- Keep in mind other **differential diagnoses** for sore throat:
  - **Viral pharyngitis (>70%)**
    - ✓ eg. infectious mononucleosis
  - Life-threatening pharyngeal infections

Wait for **positive throat culture** or **rapid antigen detection test** before initiating treatment. Goal is to prevent acute rheumatic fever, suppurative complications, & reduce symptom duration.

Pathogens

- Group A *Streptococcus* (***S. pyogenes***)

Management



GAS pharyngitis symptoms & absence of viral presentation

supports diagnosis

Throat swab

Wait for GAS culture

Positive

Negative

Penicillin V 40 mg/kg/d PO in 2 divided doses × 10 days **OR** **amoxicillin** 50 mg/kg/d PO (up to 1 g/d) in 1-2 divided doses × 10 days

Continue supportive care

March 2020
Richard He (MD Student 2022, University of Alberta) for www.pedscases.com

SUN AND INSECT SAFETY

RISK OF SUN EXPOSURE

Sunburn **Heat stroke** **Dehydration**

A sunburn is superficial skin damage that occurs when unprotected skin is exposed to the sun's ultraviolet (UV) rays. Predisposes to **skin cancer**.

SIGNS & SYMPTOMS

Sunburn: Redness or pink painful skin that is hot to touch. *Seek medical attention if blisters develop!*
Heat stroke: Headache, fever, fatigue, moist skin.
Dehydration: Stomach/leg aches, fatigue, decreased voids, light-headedness.

TREATMENT

- Stay inside or in shade, hydrate, remove excess clothing, fan your child.
- If painful, may apply cold compress or have child take cool (not cold) bath.
- Apply aloe containing gel to affected skin.
- Topical corticosteroids can reduce swelling.

5 S' of PREVENTION

Seek: Avoid sun from **11am to 3pm.** Encourage children to play in shade, under an umbrella or indoors during these hours.

Slip **Slap**: Dress children in sun-protective clothing including **wide brim hats** and **long sleeves.**

Slop: Children > 6 months should wear **SPF 30+ sunscreen** (with UVA and UVB protection). Reapply often especially when swimming or sweating.

Slide: Sunglasses **with ultraviolet protection** prevent sun damage to children's eyes.

RISKS OF INSECT BITES

- Certain insects' bites (e.g. mosquitoes and ticks) carry diseases.
- Insect bites can also cause swelling, redness, pain, itching and rarely anaphylaxis.

TREATMENT

- Symptomatic topical treatment with cold compresses, ice, and aloe are effective.
- Use oral non-sedating antihistamines, like cetirizine, for itch.
- Consider prophylaxis with anti-malarials when travelling to mosquito-endemic areas.
- **Recognize ANAPHYLAXIS**
  Most common – bees and wasps.
  Administer EpiPen and call 911.

Rapid onset, **potentially life threatening**, systemic hypersensitivity reaction

THE *DEET*AILS OF PREVENTION

- **Avoid** areas where insects breed and live, including **standing water.**
- Wear light-coloured, **long clothing and closed shoes**. Tuck shirts into pants and pants into socks.
- Apply icaridin- or DEET-containing **insect repellent** to clothing and exposed skin.

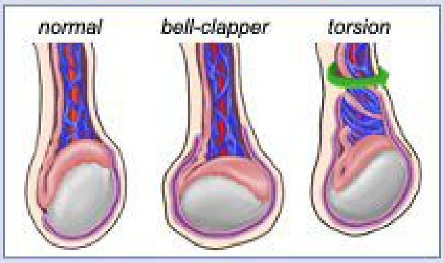
Age	How much DEET?	Icaridin?
< 6mos	NONE	NONE
6mos - 2y	10% once daily	Up to 20%
2- 12y	10% TID	Up to 20%
> 12y	Up to 30% DEET	Up to 20%

- Apply sunscreen first, then insect repellent 30 minutes later. Do not combine.

July 2023
Heather Halperin (Medical Student, University of Alberta) and Dr. L. Cynthia Gunaratnam (Pediatrician, University of Alberta) for www.pedscases.com

TESTICULAR TORSION

Intravaginal torsion: longitudinal rotation of the spermatic cord due to "bell-clapper deformity". This results in the absence of testicular blood flow and is a **surgical emergency**.



Bell-clapper deformity: Capacious scrotal sac. Often bilateral.

PRESENTATION

- **Pain**
  - Sudden onset
  - Deep / visceral
  - Unilateral
  - Testicular / scrotal
  - May radiate to inguinal or lower abdominal areas
  - Potential prior Hx of intermittent pains
- Nausea
- May have associated trauma
- Peak incidence: 12-16 y/o, unlikely before puberty
- May be awakened from sleep due to pain

PHYSICAL EXAM

Scrotum

- *Early* presentation: may be normal
- *Late*: edematous, indurated, erythematous

Affected testis

- Tender
- High riding
- Horizontal lie
- Cremasteric reflex absent

DIAGNOSIS

Primarily a **clinical diagnosis**. History and P/E often sufficient to bring straight into OR.

☐ U/A generally not indicated and not needed for diagnosis

Colour Doppler Ultrasound

If Dx is in question, U/S to determine presence or absence of blood flow:

- Decreased testicular perfusion
- Twisting of spermatic cord

Usefulness limited in small prepubertal testes with ↓baseline flow

DO NOT delay surgical management for imaging studies if clinical findings are strongly suggestive.

TWIST Score

Symptom	Points
Testicular swelling	2
Hard testicle	2
Absent cremasteric reflex	1
Nausea/vomiting	1
High riding testis	1

≤2 Points: low risk

3-4 Points: medium risk

≥5 Points: **HIGH** risk

Detorsion within…	Testis viability
4-6 hours	97-100%
>12 hours	20-61%
>24 hours	0-14%

GOAL: **early surgical consultation** with surgeon and in operating room **within 6 hours from onset of symptoms**

NEVER delay surgery on assumption of nonviability based on clinically estimated duration of torsion

≤6 hours

SURGICAL EXPLORATION

Is the torsed testicle **viable**?

- yes → Surgical detorsion and orchiopexy (fixation) → **Contralateral** testicle exploration and orchiopexy
- no → Orchiectomy (removal) → **Contralateral** testicle exploration and orchiopexy

bell-clapper often bilateral

- Average recovery time: 1-2 weeks
- One functional testicle is sufficient for normal fertility

Published May 2021
Sarah Park (Medical Student, University of Alberta), Dr. Peter Metcalfe (Pediatric Urologist, University of Alberta) and Dr. Mark Assmus (Urologist, Indiana University) for www.pedscases.com

PEDIATRIC VIRAL RASHES

*** Typical duration of rash**

*1-3 days

ROSEOLA (HHV6)

Prodrome: Abrupt, high fever, cough, coryza, pharyngitis, irritability, malaise.

Rash: Maculopapular, nonpruritic. Begins on trunk, spreads to neck and proximal extremities.

Rash onset AFTER fever subsides.

Generally < 2 years.

*~ 3 days

RUBELLA

Prodrome: Low-grade fever, pharyngitis, coryza, malaise, lymphadenopathy.

Rash: Maculopapular, +/- pruritic. Begins on face and spreads to neck, trunk, extremities. **Forchheimer spots** (pinpoint petechiae on soft palate). Incubation period: 14-28 days.Contagious period: From 1-2 weeks before appearance of rash 1-2 weeks afterwards.

Rash AND low-grade fever.

Complications: Thrombocytopenic purpura, otitis media, encephalitis. In pregnancy: congenital rubella syndrome.

*7-10 days

HAND-FOOT-and-MOUTH DISEASE (Coxsackie Virus)

Prodrome: Fever, sore throat, malaise.

Rash: Flat pink macules on palms and soles, progressing to elongated grey blisters. Painful vesicles and ulcers on buccal mucosa and pharynx. Incubation period: 3-5 days. Contagious period: most contagious during first week of illness.

Eczema coxsackium: widespread form in children with eczema.

Generally < 5 years.

*5-10 days

ERYTHEMA INFECTIOSUM (Parovirus B19)

Prodrome: Fever, malaise, +/- myalgia, +/- headache.

Rash: "Slapped cheeks" - Firm, red, hot, cheeks (lasts 2-4 days). Followed by lacy reticulated eruption over the limbs and sometimes trunk (lasts 5-10 days). Incubation period: 5-15 days. Contagious period: Begins 5-10 days following exposure and lasts about 5 days. **No longer contagious once rash is present.**

Complications: Arthritis, aplastic crisis in sickle cell disease, autoimmune hemolytic anemia, chronic infection in immunocompromised. In pregnancy: Fetal hydrops, intrauterine growth restriction, death.

*~ 7 days

MEASLES

Transmission: Airborne and droplet – **highly contagious.**

Prodrome: Fever, malaise. **The 3 C's: Cough, coryza, conjunctivitis. Koplik spots** (white/blue/grey lesions on erythematous base) on buccal mucosa.

Rash: Maculopapular, nonpruritic. Begins on face and spreads to trunk and extremities. Spreads from head down. Incubation period: 6-21 days. Contagious period: From 5 days before appearance of rash to 4 days afterward.

Rash AND high-grade fever.

Complications: Otitis media, pneumonia, croup, diarrhea, encephalitis, death.

*1-2 weeks

CHICKENPOX (Varicella Zoster Virus)

Transmission: Airborne and droplet – **highly contagious**

Prodrome: Fever, malaise, anorexia, headache.

Rash: Start as macules and progress to vesicles that crust over. +++pruritic. Begin on face, back, and stomach and progress to generalized. Lesions in different stages of healing. Incubation period: 10-21 days. Contagious period: 25-48 hours before rash erupts, lasting until lesions are crusted over.

Complications: Secondary bacterial infection, pneumonia, encephalitis, meningitis, myelitis, hepatitis, disseminated infection.

*1-2 weeks

HERPES SIMPLEX VIRUS 1

Primary infection: Gingivostomatitis - often with perioral vesicular lesions. May have fever, restlessness, excessive drooling, lymphadenopathy. Can be contagious even in the absence of symptoms.

Recurrent: Painful oral-labial vesicles on an erythematous base that crust over.

Oral lesions in neonate - consider disseminated infection.

Complications: herpetic keratitis, pharyngitis, eczema herpeticum, erythema multiforme, disseminated infection, encephalitis.

Published April 2021
Zoë Phillips (Medical Student 2021, University of Saskatchewan), Dr. Carly Rumley (Family Physician, University of Alberta), & Dr. Morgan Hewitt (General Pediatrician, University of Saskatchewan) for www.pedscases.com

TRANSIENT TACHYPNEA OF THE NEWBORN

Benign and self-limited neonatal respiratory condition characterized by rapid breathing and pulmonary edema due to delayed clearance of fetal lung fluid (FLF).

RISK FACTORS

- **Maternal:** Caesarean delivery, short/no labor, diabetes, asthma
- **Fetal:** male sex, macrosomia, SGA, prematurity

NORMAL PHYSIOLOGY

- **Secretion of FLF into alveoli:** supports lung growth & function in utero
- **Reabsorption of FLF via:**
  - Thoracic squeeze through birth canal
  - Breathing/crying
  - Activation of sodium channels

TTN PATHOPHYSIOLOGY

Delayed reabsorption of FLF
↓
Pooling in lung interstitium & interlobar fissures
↓
Decreased lung compliance
↓
Impaired lung inflation

SIGNS & SYMPTOMS

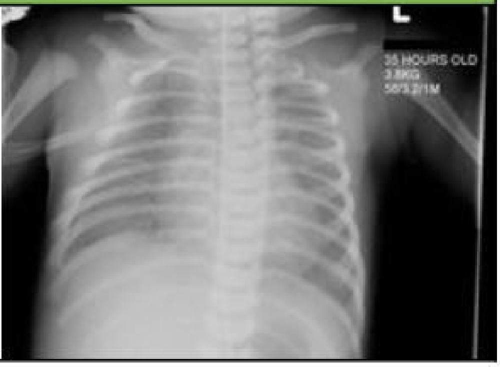
- **Timing:** immediately after birth (within first 2h)
- **Tachypnea:** RR > 60
- **Other:** cyanosis, tachycardia (HR > 160), **oxygen desaturations**
- **Inspection:** signs of increased WOB such as grunting, nasal flaring, inter/subcostal retractions
- **Auscultation:** typically, clear breath sounds, air entry may be decreased

DIAGNOSIS

- Helpful to confirm diagnosis with chest x-ray and blood gas if severe
- Symptoms should resolve within 12-72h

INVESTIGATIONS

- **Chest XR:** perihilar streaking, hyperinflation, fluid in interlobar fissures
- **Blood Gas:** assess for respiratory acidosis
- Extended workup for differential diagnoses if clinically indicated



DIFFERENTIAL DIAGNOSES

Respiratory distress syndrome, meconium aspiration syndrome, pneumothorax, pneumonia, persistent pulmonary hypertension, sepsis, congenital heart disease, CNS disease, inborn errors of metabolism

MANAGEMENT

- **Supportive Care:**
  - Keep warm
  - NPO
  - IV fluids if RR > 80 and increased WOB
  - Enteral tube feeds if stable and RR < 80
- **Respiratory Support (if indicated):**
  - Nasal CPAP to relieve severe WOB
  - Supplemental oxygen if hypoxemic, target SpO2 of 92-96%, FiO2 usually <40%

October 2024
Nikki Nguyen (Resident, University of Calgary), Dr. Jason R. McConnery (Pediatric Respirologist, McMaster University) for www.pedscases.com

TURNER SYNDROME

- **~1/2500 live births**
- Complete or partial loss of X chromosome → 45,X
- Can occur w/ mosaicism

RISK FACTORS

- No established risk factors
- No link to maternal age
- Recurrence is rare

DIAGNOSIS

PRE-NATAL

- Standard prenatal screening does not accurately detect
- Suggestive US findings
  - Cystic hygroma
  - Hydrops
  - Lymphedema
  - Aortic coarctation
- Karyotype via CVS/amnio

INFANCY / CHILDHOOD

- Karyotype if suspected
- Infancy
  - ≥ 2 dysmorphic features
- Childhood
  - Short stature
  - No breast development
  - Amenorrhea
  - Infertility

PRESENTATION

CLASSIC FEATURES

- **Neonatal:** redundant nuchal skin, hand/foot lymphedema
- **Childhood:** short stature, ↓ growth velocity
- **Adolescent**: delayed puberty, lack of breast development, primary amenorrhea (↑ FSH)
- **General**
  - **Head:** webbed neck, low posterior hairline
  - **Ears:** posteriorly-rotated, looped helices
  - **Mouth:** malocclusion, micrognathia
  - **Body:** broad chest, widely-spaced nipples
  - **Extremities:** cubitus valgus, hyperconvex nails, short 4th metacarpals

COMPLICATIONS

- **OPT:** strabismus, congenital glaucoma
- **HEENT:** SN hearing loss, recurrent AOM
- **DENT:** orthodontic anomalies (e.g. malocclusion)
- **CV:** cardiac anomaly (33%), hypertension
  - Bicuspid aortic valve > aortic coarctation > progressive aortic root dilatation/dissection
- **GI:** transaminitis, Celiac, IBD
- **GU:** gonadal dysgenesis, renal malformation
  - Horseshoe kidney > duplicated/cleft renal pelvis
- **MSK:** congenital hip dysplasia, scoliosis, JIA
- **CNS:** developmental delay, learning disabilities
- **ENDO:** short stature, hypothyroid, T2DM, obesity
- **DERM:** keloid formation, pigmented nevi

MANAGEMENT

SURVEILLANCE

- Consult genetics + peds endo, use TS growth charts
- Refer to relevant clinical practice guidelines for details
- **OPT:** consult ophtho by 2yo, then yearly f/u
- **HEENT:** hearing test in childhood + adolescence
- **CV:** newborn echo + peds cardio consult, monitor BP/pulses/murmurs, repeat consult in adolescence
- **GI:** celiac screening + liver enzymes
- **GU:** renal US at Dx, repeat if recurrent UTIs/HTN
- **MSK:** observe for hip dysplasia + scoliosis
- **ENDO:** monitor puberty, thyroid, HbA1C, vitamin D

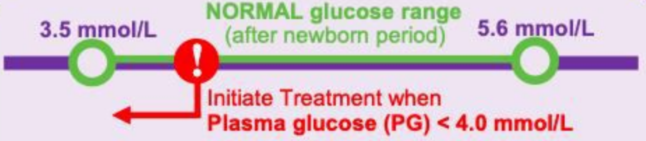
TREATMENT

- **Short Stature:** Growth Hormone → start in early childhood if height < 5th %ile + poor growth velocity
- **Gonadal Dysgenesis:** if Y-chromosome material present → prophylactic gonadectomy to ↓ risk of gonadoblastoma
- **Ovarian Failure:** start Estrogen by 13yo if no pubertal development, add Cyclic Progesterone after 2y, counsel regarding fertility
- **Lymphedema:** garments, exercises, diuretics
  - May be exacerbated by Estrogen

August 2022
Summer Hudson (Medical Student, University of Alberta), Dr. Marian Thorpe (Pediatric Endocrinology Fellow, University of Alberta), & Dr. Melanie Lewis (General Pediatrician, Stollery Children's) for www.pedscases.com

TYPE 1 DIABETES MELLITUS: OVERVIEW

- **Pathophysiology:** **Autoimmune** destruction of insulin-producing **pancreatic beta cells** in the islets of Langerhans, resulting in **insulin deficiency**
- **Age of presentation:** Can present at any age; Bimodal distribution of peaks at 4 - 6 years old and early puberty (10 – 14 years old)
- **Risk factors:** both genetic (relatives with T1DM) and environmental factors
  - In **genetically susceptible individuals**, exposure to one or more **environmental agents** likely triggers **immune response**



RECOMMENDED GLYCEMIC TARGETS

- ✓ **A1C ≤ 7.5** (7 in some guidelines) **fasting** PG **4.0 – 8.0** mmol/L, **postprandial** PG **5.0 – 10.0** mmol/L
- ✓ Pre-prandial targets of 6.0-10.0 mmol/L
- ✓ Consider higher A1C target if prior significant hypoglycemia or hypoglycemia unawareness

DIAGNOSIS

Clinical Presentation	Investigations
▪ **Polyuria:** osmotic diuresis due to increased urinary glucose excretion • Secondary **enuresis** or **nocturia** in children ▪ **Polydipsia:** result of increased serum osmolality ▪ **Polyphagia** ▪ **Diabetic Keto Acidosis (DKA)** ▪ **Weight loss:** due to hypovolemia, increased catabolism (impaired glucose utilization, increased muscle & fat breakdown) ▪ Factors that may help differentiate T1DM vs T2DM : • *Body habitus:* Obesity is important risk factor for T2DM	☐ **Diagnosis of diabetes:** ▪ **Fasting** PG **≥ 7 mmol/L** ▪ **Random** PG **≥ 11.1 mmol/L** ▪ **PG ≥ 11.1** 2 hr post 75 g OGTT (OGTT not frequently used for diagnosis in children) ▪ **A1C not used** to diagnose T1DM in children ☐ If asymptomatic, second test always needed; single test sufficient if symptomatic ☐ Lab results to support T1DM vs T2DM: antibodies (e.g., GAD65), insulin, C-peptide

MANAGEMENT

Education	Insulin
✓ Training for patients & families by **multidisciplinary teams:** ▪ *Insulin; BG & ketone monitoring; sick-day management; DKA; hypoglycemia; correction factors* ✓ Anticipatory guidance ✓ Dietician referral ✓ **Carbohydrate counting** to match insulin to intake, allows flexibility in diet (I:C ratio)	➢ **Treat to meet glycemic targets** ➢ Two types of intensive insulin regimens: **Multiple daily injections** (MDI) & **continuous subcutaneous insulin infusion** (CSII; insulin pump) ➢ Less intensive BID & TID regimens ➢ Fingerstick or continuous glucose monitoring (CGM) ➢ **Hypoglycemia management:** • Mild & moderate: oral simple carbohydrate e.g., **fruit juice** • Severe: **glucagon** (IM, intranasal, SC), **IV dextrose**

ACUTE COMPLICATIONS

- ! **Hypoglycemia** (due to insulin)
- ! **Diabetic Ketoacidosis (DKA)**

SCREENING

Check for…	Starting…	Frequency
Glycemic control (**A1C**)	At Dx	q3months
Autoimmune thyroid (**TSH & TPO**)	At Dx	q2years
Celiac & Adrenal insufficiency	-	As indicated
Nephropathy	Age **12** (if Dx >5 years ago)	Annually
Retinopathy	Age **15** (if Dx >5 years ago)	Annually
Neuropathy	Age **15** (with poor glycemic control)	Annually (after 5 years of Dx)
Dyslipidemia	Age **12** (if no extra risk factors)	Repeat at 17
Hypertension	At Dx	q6months

LONG TERM COMPLICATIONS

Microvascular Disease	Macrovascular Disease	
• Diabetic nephropathy • Diabetic retinopathy • Peripheral & autonomic neuropathy	• Cardiac disease (CAD, MI) • Peripheral vascular disease • Cerebrovascular disease (stroke)	**Hypertension** **Dyslipidemia**

Published July 2021
Alyssa Kahane (Medical Student, University of Ottawa) and Dr. Alexa Marr (Pediatric Endocrinologist, Children's Hospital of Eastern Ontario) for www.pedscases.com

URINARY TRACT INFECTION (UTI)

Key Points

- **Infants & young children** → non-specific presentation:
  - **Fever** & **vomiting** most common
- **Verbal children** → fever, abdominal pain, & **urinary symptoms** common:
  - Dysuria
  - Urgency, frequency, & new incontinence
  - Hematuria

Urinalysis supports diagnosis:
- ✓ **Toilet-trained:** clean catch urine
- ✓ **Not toilet-trained:** in-and-out catheter

↑ **leukocyte esterase, nitrites,** or **WBCs** on microscopy suggests UTI. Confirm by **urine culture**.

Pathogens

- ***Escherichia coli*** (80% of pediatric UTIs)
- Other enteric Gram negative organisms (eg. *Klebsiella pneumoniae*)

Management

Empiric **3rd-gen. cephalosporin** (less *E. coli* resistance)
→ eg. **cefixime** 8 mg/kg/d PO daily

If not tolerating PO intake or <3 months old, consider:
IV antibiotics *(eg. ceftriaxone or gentamicin)*
Narrow antibiotics based on sensitivities

Afebrile, immunocompetent → 3-5-day course

Febrile → 7-10-day course

Perform renal U/S if:
1st febrile UTI in children <2 years to assess renal/bladder anatomy & risks for recurrent UTI.

Early intervention key in preventing renal damage.
Can start empiric antibiotic therapy ***immediately*** after sterile urine collection if clinical/lab suspicion high.

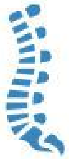
March 2020
Richard He (MD Student 2022, University of Alberta) for www.pedscases.com

VACTERL ASSOCIATION

A group of anomalies that occur in the newborn
- Approximately **1 in 10,000 to 40,000 births**
- The cause of VACTERL association is **unknown**
- The mode of inheritance is **sporadic**
- Greater frequency in **infants of diabetic mothers**

DIAGNOSIS

- VACTERL is a **clinical diagnosis**
- **≥ 3 features** required for diagnosis
- **Diagnosis of exclusion** – other possible causes of birth defects need to be ruled out

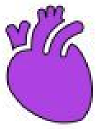


Vertebral defects

- Present in 60-80% of cases
- Includes hemivertebrae, hypoplastic vertebrae, extra or missing vertebrae, congenital scoliosis, spina bifida
- Can cause back pain later in life

Anal atresia

- Present in 60-90% of cases
- Narrowing or lack of patency of the anus
- May be accompanied by genitourinary abnormalities
- No passage of meconium, abdominal distension, vomiting



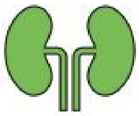
Cardiac defects

- Present in 40-80% of cases
- Subtle to life threatening defects
- Common defects: ventricular septal defect, atrial septal defect, tetralogy of fallot, patent ductus arteriosus

Tracheo-esophageal fistula

Esophageal atresia

- Present in 50-80% of cases
- TEF is an abnormal connection between the trachea and the esophagus
- EA is a congenital condition characterized by the incomplete development of the esophagus
- TEFs are most often found in conjunction with EA
- May present with vomiting, copious oral secretions, feed intolerance, coughing, cyanosis, or respiratory distress



Renal anomalies

- Present in 50-80% of cases
- Renal aplasia, renal dysplasia, vesicoureteral reflux, displaced or mal-positioned kidneys
- May not cause problems immediately
- Nephrology and urology consultation may be necessary

Limb abnormalities

- Present in 50% of cases
- Radial aplasia – underdevelopment of the radius
- Polydactyly (additional digits), missing digits, lower limb hypoplasia, syndactyly (webbing)

A **syndrome** involves a collection of signs and symptoms that consistently occur together due to an underlying cause, while an **association** refers to a statistical tendency for certain features to co-occur without an underlying cause.

MANAGEMENT

- Treatment is directed toward the **specific malformations and related symptoms**, which varies greatly.
- Many of the structural abnormalities (heart defects, anal atresia, TE fistula, radial defects, etc.) can be **surgically repaired**. Some surgeries occur in the neonatal period, while others occur later in life.
- Repeat surgeries might also be needed to further correct structural defects.

September 2024

Katharine V. Jensen (Pediatrics Resident, University of Alberta) and Dr. Karen Forbes (Professor of Pediatrics, University of Alberta) for www.pedscases.com

VARICELLA

Clinical Presentation

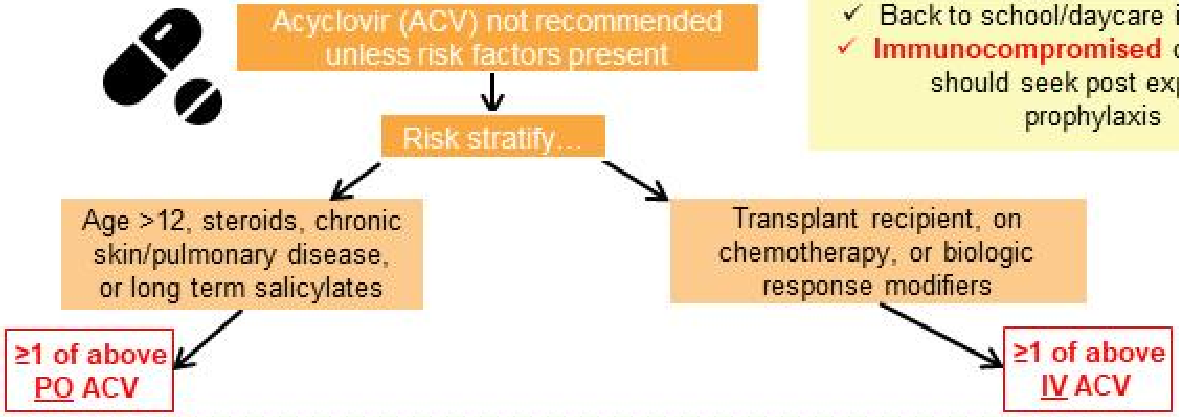
- **Prodrome**: fever, malaise, anorexia, headache
- Rash is widespread, pruritic rash, erythematous macules which progress to blisters that crust over
- Rash is not uniform, lesions at **different stages of healing**, centripetal distribution
- Most common complication is secondary bacterial infections from **group A streptococcus, Staphylococcus aureus**
- Reactivation of latent infection decades later resulting in **Herpes Zoster (Shingles).** Rare in healthy children, often **history of chickenpox or vaccine**, rapid recovery.

Pathogenesis

- **Transmission**: respiratory or oral secretions
- Stays **airborne** for many hours, may be acquired from being in the same room as an infected person
- 1st viremia (prodrome symptoms), 2nd viremia (widespread rash)
- **Breakthrough varicella**: mild form (<50 lesions), most often after only single dose of vaccine
- Replicates in lymphoid tissue. Virus remains latent for life in a **dorsal root ganglion**
- Virus can **reactivate** at any point, especially if the person becomes **immunosuppressed**

Management

- Most sensitive test: **PCR** from skin lesions



Acyclovir (ACV) not recommended unless risk factors present

Risk stratify…

Age >12, steroids, chronic skin/pulmonary disease, or long term salicylates

≥1 of above PO ACV

Transplant recipient, on chemotherapy, or biologic response modifiers

≥1 of above IV ACV

When can they go back to school?

✓ Back to school/daycare if feels well

✓ **Immunocompromised** classmates should seek post exposure prophylaxis

Prevention

✓ Prevention through **vaccination**

- Live Attenuated Vaccine given SubQ in **2 doses** (12 months then at 18 months or 4 years)
- **Contraindicated** if: previous anaphylactic reaction, pregnancy, immunosuppressive therapy, leukemia/lymphoma

Who gets a post exposure prophylaxis (VZIG)?

1. Immunosuppressed patients (VZIG but may use vaccine in lower risk cases).
2. Premature babies (<28 weeks).
3. Newborns exposed to varicella (5 day pre-delivery to 2 day post-delivery).
4. Pregnant women without evidence of immunity to varicella.

** Note on neonatal varicella: consult infectious disease. Management is complicated.*

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Aryan Riahi (MD Student, University of British Columbia) &
Dr. Joan Robinson and Dr. Gauri Shah, Pediatric Infectious Diseases for pedcases.com

VESICOURETERAL REFLUX

Vesicoureteral Reflux (VUR): Retrograde urine flow from bladder into the upper urinary tract (UT). It is a structural or functional abnormality that can predispose patients to renal scarring in the setting of UTI.

CLINICAL RELEVANCE

While not always clinically significant, VUR accompanied by UTIs, can result in **renal scarring** and **potentially progress to reflux nephropathy.**

PRESENTATION

- May be asymptomatic or have lower/upper UTI (non-specific symptoms & can be hard to elicit in infants; consider fever, irritability, dysuria, etc.)
- VUR may be identified after investigation of antenatal hydronephrosis or febrile UTIs

HISTORY

- **Recurrent febrile or non-febrile UTI**
- **Family history of VUR**
- Bladder/bowel dysfunction
- Hydronephrosis (found incidentally on pre or postnatal renal U/S, raising suspicion for VUR)

DIAGNOSIS

Voiding Cystourethrogram (VCUG)

- Gold standard for diagnosing/grading VUR
- Utilizes urethral & bladder fluoroscopy with contrast
- Allows direct visualization of UT anatomy & urine flow directionality
- Requires catheter insertion, can be uncomfortable for kids

PRIMARY VUR MANAGEMENT

Based on presentation severity, age, extent of renal scarring, and VUR grade.

Conservative Approach

Prompt treatment of culture proven UTIs based on sensitivity. Low grade VUR often spontaneously resolves.

Antibiotic Prophylaxis

Used in the setting of recurrent UTI or high-risk patients with concerns of renal scarring. TMP-SMX/nitrofurantoin commonly chosen. Amoxicillin for 2 months old or less. Duration should be limited due potential risks including resistant organisms and medication side effects.

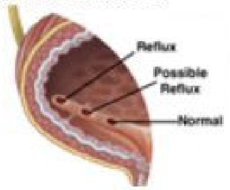
Endoscopic or Open Surgical Correction

Reserved for patients with unresolved VUR experiencing recurrent UTI after failing antibiotic prophylaxis.

PATHOPHYSIOLOGY

PRIMARY VUR

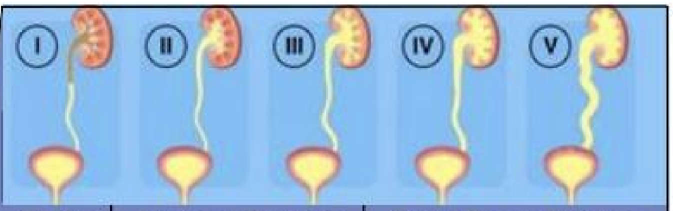
Inadequate closure of the ureterovesical junction (UVJ), due to a short intravesical ureter



SECONDARY VUR

High voiding pressure in the bladder resulting in failure of UVJ closure during bladder contraction

Examples include:
- Posterior Urethral Valves
- Neurogenic Bladder
- Bowel/Bladder Dysfunction



Grade	Ureteral Dilation	Renal Pelvis Dilation
I	None	None
II	None	None
III	Mild/Moderate	Moderate
IV	Moderate	Moderate
V	Gross with kinking	Severe

* Orange boxes indicate extent of urine reflux

Antibiotic Treatment of UTI

1. Empiric Tx if positive urinalysis & UTI symptoms.
2. Then tailer choice based on culture/sensitivities.

PO: TMP-SMX, 2nd/3rd generation cephalosporins (eg. Cefixime) or amoxicillin-clavulanate

IV*: 3rd generation cephalosporins (eg. Ceftriaxone)

*for example: bacteraemic/septic patients, age < 2 months, unable to tolerate to oral abx, etc.

SECONDARY VUR MANAGEMENT

Treat underlying cause. Anatomic abnormalities may be treated surgically. Patients may require intermittent catheterization to treat retention and reduce bladder pressures.

October 2024
Neel Phaterpekar (Medical Student, University of Alberta), Dr. Darcie Kiddoo (Pediatric Urologist, University of Alberta) for www.pedscases.com

VON WILLEBRAND DISEASE

Von Willebrand Disease (vWD) is caused by a decreased level of functioning **von Willebrand factor**, which plays a crucial role in primary hemostasis. Most forms of vWD follow an inherited **autosomal dominant** pattern that affects males and females equally. vWD is the most common inherited bleeding disorder and affects approximately **1% of the population** but only 1% of those with vWD have symptoms.

PATHOPHYSIOLOGY

- Qualitative defect or quantitative deficiency of vWF
- vWF needed for platelet adhesion and aggregation, and acts as a chaperone for Factor VIII, extending its half life in circulation. Thus, vWD affects both primary and secondary hemostasis
- vWF exists as a series of multimers ranging in size, with the largest being the most active in mediating platelet adhesion/aggregation
- vWF levels vary according to blood group (non-group patient patients have higher levels)

PRESENTATION

Excessive and prolonged bleeding:

- Frequent nosebleeds
- Oral mucosa bleeding
- GI bleeding
- Heavy menstrual cycles
- Easy bruising
- Prolonged bleeding after surgery or trauma

Can use the Pediatric Bleeding Questionnaire (PBQ) to assess bleeding.

DIFFERENTIAL DIAGNOSIS

- Hemophilia A, B, C
- Bernard Soulier syndrome
- Platelet disorders
- Antithrombotic therapies
- Trauma: accidental or inflicted

CLASSIFICATION

Type 1	**Mild quantitative defect** (decreased amount of vWF and proportional decrease in vWF activity)	80% of cases
Type 2	**Qualitative defect** (vWF activity disproportionally lower than quantity)	20% of cases
Type 3	**Severe total quantitative defect** (no vWF produced)	1 per million

INVESTIGATIONS

Personal and family history of bleeding with consistent labs:

- CBC, platelets, PTT
- Plasma vWF antigen levels
- vWF:Ristocetin cofactor activity (determine how well vWF binds platelets)
- Factor 8 levels

If these are abnormal consider further tests to determine subtype:

- vWF multimer distribution with gel electrophoresis
- Ristocetin-induced platelet aggregation test

TEST RESULTS

Test	Expected Result
PTT	Normal or increased
Factor VIII	Normal or decreased
Platelet Count	Normal or decreased
Ristocetin Activity	Decreased (cofactor for vWF-platelet binding)
von Willebrand antigen	Decreased
Blood group	Decreased vWF antigen in group O
vWF multimer analysis	Multimer variants

MANAGEMENT

Supportive:

- Stop the bleeding
- Ensure **hemodynamic stability**
- **Minimize trauma**
- Avoid **medications** that could worsen bleeding symptoms
- Only ~10% require long-term prophylactic therapy

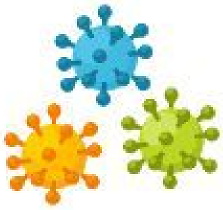
Medical management:

- **Desmopressin** (ADH analogue): a long-term therapy shown to increase vWF and factor VIII
- **vWF replacement therapy**: severe deficiency, bleeding, or short-term prophylaxis for surgery
- **Estrogen therapy**: menorrhagia
- **Tranexamic acid:** dental procedure prophylaxis

Published July 2022
Morgan Gregg (Medical Student, University of Alberta), Dr. Mia Lang (Pediatrician, University of Alberta) for www.pedscases.com

VERRUCA VULGARIS (WARTS)

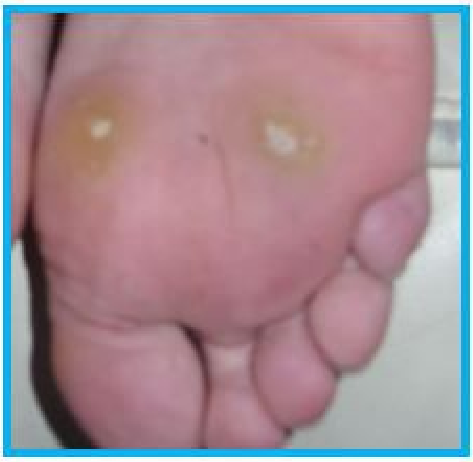
PATHOGENESIS



- **Human Papillomavirus (HPV)** is the causative agent.
- HPV **infects the basal keratinocytes** in the epithelium.
- **Epithelial hyperplasia** leads to the classic appearance.
- Transmission through **skin-to-skin contact** and **surfaces**

CLINICAL PRESENTATION

- **Prevalence** peaks during **adolescence**.
- Appear as hyperkeratotic, exophytic papules.
- Commonly on **dorsal aspect of the hands/fingers** and plantar surface of feet.
- Can have **thrombosed capillaries** and **interruption of the skin lines.**



MANAGEMENT

- **Watchful waiting** (>75% of cases **self-resolve** by 2 years).

1 **First line**

- Salicylic acid
- Cryotherapy

2 **Second line**

- Cantharidin*

3 **Third line**

- Bleomycin
- Laser therapy
- Intralesional immunotherapy

* Can cause a circle of warts around the lesion called a donut wart.

PREVENTION

- ✓ Patients can spread the virus to an unaffected area of skin. Therefore, inform patients **not to touch their warts** and to groom the affected body part with different equipment to prevent further spread (e.g. do not share pumice stones with family members).
- ✓ HPV vaccine only prevents genital warts.

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